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Attachment 3

Toxicity

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D4-3-1. INTRODUCTION

This section contains summaries on the information used in determining the TRVs for the inorganic contaminants for which toxicological studies were located.

D4-3-1.1 1,1 Dichloroethylene (CAS No. 75-34-4)

Groups of 48 each male and female Sprague-Dawley rats (Spartan substrain) were administered 50, 100, or 200 ppm 1,1-dichloroethylene in drinking water for a period of 2 years. Control groups of 80 animals/sex were maintained for the same period. Daily intake was calculated to be 7, 10, or 20 mg/kg bw/day for males and 9, 14, or 30 mg/kg bw/day for females. There were no treatment-related effects on mortality, body or organ weight, clinical chemistry, urinalysis, hematology, or numbers of tumors. The only pathologic findings were of hepatic lesions, generally characterized by minimal mid-zonal hepatocellular fatty change and hepatocellular swelling. These findings were noted in rats of all female treatment groups. In male rats, only the 200 ppm treatment group showed a statistically significant increase in the incidence of hepatocellular swelling, but this trend was also observed in animals receiving 100 ppm 1,1-dichloroethylene.

As part of this same study, beagle dogs (4/sex/group) were administered 6.25, 12.5, or 25 mg/kg bw/day in gelatin capsules. Treatment for 97 days had no effect.

This study, and a review of the available data, indicate that the liver is the most sensitive target organ and, furthermore, that rats are the most sensitive species. It is, therefore, appropriate to set an RfD based on the LOAEL of 9 mg/kg bw/day for hepatic lesions in female rats.

In inhalation studies, both sexes of Swiss mice were exposed to 10 and 25 ppm (MTD) for 4-5 days/week for 12 months. A statistically significant increase in kidney adenocarcinoma was noted in male mice. Although statistically significant increases in mammary carcinomas in female mice and pulmonary adenomas in both sexes were reported, dose-response relationships were unclear. A second study exposed Sprague-Dawley rats to 10, 25, 50, 100, or 150 ppm, 4-5 days/week for 12 months and observed them until spontaneous death. A statistically significant increase in total mammary tumors, but not carcinomas alone, was seen only at 10 and 100 ppm (IRIS 1994).

1,1-Dichloroethylene has been shown to be fetotoxic, but not teratogenic to rodents after exposure in drinking water or by inhalation.

D4-3-1.2 1,1,1-trichloroethane (CAS 71-55-6)

A multigenerational study was modified to include screening for dominant lethal and teratogenic effects for drinking water exposure to both male and female ICR Swiss mice (Lane et al. 1982). Three dose levels were studied: 100, 300 and 1,000 mg/kg-d. No significant differences were observed at any dose level and the study did include two-generations. Therefore the maximum dose level is considered to

be a NOAEL. In addition, the National Cancer Institute (NCI 1977) conducted a two year study to determine if there are any carcinogenic properties to 1,1,1-trichloroethane from ingestion. Based on the results of this study a NOAEL of 5,615 mg/kg-d was established. A dose level of 10,000 mg/kg-d administered to mice for 6 weeks also did not show any adverse effects.

However, a single dose of 11,240 mg/kg-d administered orally to female mice has been shown to be a LD_{50} (Verschueren, 1983). Other LD_{50} are 5,660 mg/kg-d for female rabbits and 9,470 mg/kg-d for male guinea pigs (Verschueren, 1983).

The majority of the toxicity of 1,1,1-trichloroethane is attributed to its inhalation. Inhalation of 1,1,1-trichloroethane depresses the central nervous system and causes irritation and inflammation of the lungs with the effects becoming more obvious at higher doses (Torkelson et al. 1958; IRIS 1994).

D4-3-1.3 1,1,2,2-Tetrachloroethane (Freon 113) (CAS No. 79-34-5)

1,1,2,2-Tetrachloroethane is considered one of the more toxic of the simple chlorinated hydrocarbons (Clayton and Clayton 1981). Target organs include the liver, kidney, and CNS. It causes hepatocellular cancer in rats and mice exposed via gavage (NCI 1978) and has been shown to cause fetal malformations and embryotoxic effects in mice (Schmidt 1976).

In a bioassay undertaken by NCI (1978) 50 each male and female Osborne—Mendel rats and B6C3F1 mice were gavaged with technical grade (90% pure) 1,1,2,2,-tetrachloroethane in corn oil, 5 days/week. Treatment was over 78 weeks, followed by observation periods of 32 weeks for the rats and 12 weeks for the mice. The high and low average doses (incorporating varying dosage levels throughout the treatment period) were, respectively, 108 and 62 mg/kg/day for male rats, 76 and 43 mg/kg/day for female rats, and 282 and 142 mg/kg/day for mice of both sexes. Control groups consisted of 20 animals/sex and species. Vehicle controls received corn oil at the same rate as the high-dose animals; untreated controls were not intubated. Ten of the high-dose female rats died within the first 5 weeks of the study, but the association between increased dosage and elevated mortality was not statistically significant for male rats. Significantly increased mortality was also evident in the high-dose mice of both sexes. No statistically significant incidence of neoplasms was observed in rats. A highly significant dose-related increase in the incidence of hepatocellular carcinomas was observed in both male and female mice (IRIS 1994).

D4-3-1.4 1,2,4-Trichlorobenzene (CAS No. 120-82-1)

No adequate studies on the carcinogenicity of 1,2,4-trichlorobenzene are available. Acute oral LD_{50} values of 756 and 766 mg/kg have been reported for rats and mice, respectively (Sax 1984). Few long-term studies on the effects of chronic exposure to 1,2,4-trichlorobenzene have been conducted. The available data show that slight changes in liver, kidney, and adrenal glands. No exposure-related effects were observed in rats, rabbits, and monkeys exposed to 25, 50, or 100 ppm 1,2,4-trichlorobenzene seven hours a day for 26 weeks (Coate et al. 1977). Robinson et al. (1981) reported adrenal gland enlargement in male and female rats of two generations continuously exposed to 400 ppm 1,2,4-trichlorobenzene in drinking water for 95 days. No adverse effects were observed in animals to 100 ppm in drinking water.

A study by Carlson and Tardiff (1976) where 1,2,4-Trichlorobenzene was administered to rats concluded that 1,2,4-Trichlorobenzene can induce the metabolism of foreign organic compounds.

At birth of the F0 generation, litters (17-23 litters/dose group) were randomly reduced to 4 males and 4 females. Male and female progeny were dosed with 0, 25, 100 or 400 ppm of 1,2,4-trichlorobenzene (TCB) in the drinking water. The study ended when the F2 generation was 32 days old. Fertility (as indexed by conception rate of dams) of the F0 and F1 generation rats was not affected by treatment. A LOAEL was derived from a significant increase (11% in males, 13% in females) in adrenal gland weights observed in the 400-ppm groups of males and females of the F0 and F1 generations.

Watanabe et al. (1977) reported the results of a subchronic inhalation study in which rats were exposed to TCB for 6 hours/day, 5 days/week for 90 days. The exposure concentrations tested were 0, 3 and 10 ppm (0, 22.3 and 74.2 mg/m^3). The results showed a very weak sporadic increase in urinary porphyrins at 10 ppm. The 10-ppm exposure was considered a LOAEL and 3 ppm was considered a NOAEL.

In the study reported by Kociba et al. (1981), male rats, rabbits and dogs were exposed to 0, 30 or 100 ppm (0, 223 or 742 mg/m^3) for 44 days. No significant effects were observed for body weight gain, hematologic parameters, serum biochemical tests or microscopic appearance of tissues. A reversible increase in urinary excretion of porphyrins was noted. The authors interpreted this change as a compound-specific physiologic effect rather than a sign of toxicity.

Kitchin and Ebron (1980) examined the maternal reproductive and hepatic effects of TCB upon CD-1 rats that were dosed with 0, 36, 120, 360 or 1200 mg/kg/day of TCB on days 9-13 of gestation. Among the treatment groups of 9 dams/group, alteration of embryonic parameters was noted only in the 360 mg/kg/day group. (All of the dams in the 1200 mg/kg/day group died.) The observed changes included significant retardation of all four growth criteria (i.e., head length, crown-rump length, somite number, and protein content). TCB did not cause increased resorptions, embryolethality or teratogenicity. This study also demonstrated significantly increased xenobiotic hepatic enzyme activity at 120 and 360 mg/kg/day (IRIS 1994).

D4-3-1.5 1,4-Dichlorobenzene (CAS No. 106-46-7)

In a two-generation reproductive study Sprague-Dawley rats (P1) {28/sex/group} were exposed to 1,4-dichlorobenzene (1,4-DCB) vapor at concentrations of 0, 50, 150, or 450 ppm (0, 301, 902, 2705 mg/m^3) for 10 weeks, 6 hours/day, 7 days/week, then the rats were mated for 3 weeks. For exposure of the next generation, selected F1 weanlings were exposed to 1,4-DCB for 11 weeks then mated. Adult males in the 150 ppm group exhibited reduced body weights and weight gain, reduced food consumption, increased incidence of tremors, unkempt appearance and nasal and ocular discharges. At 450 ppm there was a decrease in live births, a decrease in pup weights, and decreased pup survival at day 4 of lactation for both the F1 and F2 generations. In addition, histological observations showed significant increases in incidence of hepatocellular hypertrophy in F0 and F1 males and females. No developmental abnormalities were observed in the pups examined.

The NOAEL established from this study was 50 ppm (301 mg/cu m) and the LOAEL is 150 ppm (902 mg/m^3); the critical effect was the significant increase in liver weights of P1, parental males.

An NTP (1987) chronic bioassay study was performed for 1,4-DCB in which 50 male and female F344 rats and 50 male and female B6C3F1 mice were assigned to each dose group. Female rats and both sexes of mice received 0, 300 and 600 mg/kg and male rats received 0, 150 and 300 mg/kg-day of 1,4-DCB in corn oil gavage for 2 years. An increased incidence of nephropathy was noted in low-dose and high-dose female rats when compared with vehicle controls (43, 64 and 84% incidence in control, low

and high-dose groups, respectively). There were no other significant dose-related lesions noted in female rats. From the results noted in female rats a LOAEL of 300 mg/kg-day was determined.

Hayes et al. (1985) exposed rabbits to 0, 100, 300 or 800 ppm (0, 601, 1804 or 4810 mg/m^3) for 6 hours/day on days 6–18 of gestation. Twenty-four to 28 dams and their litters were examined at the various dose levels. The maternal body weight gain during gestation as well as absolute and relative liver and kidney weights were also determined. The number of litters, corpora lutea/dam, implantation sites/dam, fetuses/litter, resorptions/litter, fetal sex ratio, fetal body weights and fetal crown-rump lengths were determined. Only the differences in percentage of implantations resorbed and percentage of litters with resorptions for the 300 ppm group were statistically significant. The occurrence of retroesophageal positioning of the right subclavian artery was increased in the 800 ppm group and was determined to be not indicative of a teratogenic response. The authors concluded that no significant teratogenic or fetotoxic effects were observed at 100, 300 or 800 ppm (IRIS 1994).

D4-3-1.6 2-Butanone (Methyl ethyl ketone, MEK) (CAS No. 78-93-3)

Cox et al. (1975) conducted a multigenerational study with measurement of development toxicity endpoints in rats. Male weanling rats were given an average of 0, 538, 1,644, 5,089 mg/kg-day 2-butanol (2-butanone's primary metabolite) in drinking water, while females were given an average of 0, 594, 1,771, and 4,571 mg/kg-day. After 8 weeks exposure, males and females were mated. F1 generations showed decreased number of live births, decreased number of pups per liter, decreased mean body weight per pup, and increased number of developmental effects in pups born to animals in the high dose group. Therefore, based on these development effects, a LOAEL of 3,122 a NOAEL of 1,771 mg/kg-day was established.

Sprague-Dawley rats were exposed to 0, 1,126, or 2,618 ppm MEK (0, 3,320, or 7,720 mg/m^3, respectively, assuming 25°C and 760 mm Hg) 7 hours/day during gestational days 6-15 (Schwetz et al. 1974). No maternal effects or effect on the incidence of fetal resorptions were observed. A decrease in fetal body weight and crown-rump length were observed in the 1,126 ppm offspring, however, these effects were not observed in the offspring of the rats exposed to 2,618 ppm. There were no gross, soft tissue, or specific skeletal anomalies that occurred at a significantly increased incidence among litters of dams exposed to 1,126 ppm MEK. However, the total number of litters containing fetuses with anomalous skeletons was significantly increased compared with controls. In the fetuses of the 2,618-ppm group, there was a significantly increased number of fetuses and litters having gross anomalies (two acaudate fetuses with an imperforate anus and 2 brachygnathous fetuses).

Pregnant Swiss mice were exposed to 0, 398, 1,010 or 3,020 ppm methyl ethyl ketone (0, 1,174, 2,978 or 8,906 mg/m^3, respectively, assuming 25°C and 760 mm Hg) 7 hours/day during gestational days 6-15. The number of dams exposed to methyl ethyl ketone ranged from 23 to 28 mice depending upon exposure group. The only maternal effect observed was a concentration-related increase in relative liver and kidney weight. A decrease in fetal body weight was also observed in the 3,020 ppm exposed group. Additionally, although no significant increase of any single malformation was found, there were several malformations (cleft palate, fused ribs, missing vertebrae and syndactyly) present at low incidences in exposed groups. Neither maternal nor developmental toxicity was observed at exposures at or less than 1,010 ppm. At 3,020 ppm, an equivocal maternal effect was reported; however, mild developmental effects (decreased fetal body weight and misaligned sternebrae) were found. Based on the absence of both maternal and developmental toxic effects, a NOAEL of 1,126 ppm is established. The LOAEL is established at 3,020 ppm (IRIS 1994).

No toxicological data describing potential adverse effects from avian exposure to 2-butanone were available.

D4-3-1.7 2-Chlorotoluene (CAS No. 108-41-8)

Weanling rats (20 each sex per treatment group) were given aqueous solution containing 2-chlorotoluene by gavage Gibson et al. (1974a). Doses were 0, 20, 80, or 320 mg/kg-day for 103 days. Male rates in the 80 and 32 mg/kg-day groups had statistically lower mean body weight than controls and an increase in adrenal gland weight. Males in the 320 mg/kg-day group had increased heart and tests weights and an increased white blood cell count. Therefore, based on this study, a NOAEL of 20 mg/kg-day was established. A total AF of 4 was applied to this NOAEL to yield a TRV of 5 mg/kg-day.

In the absence of data for mammalian herbivores, the Gibson et al. (1975) study was used. In this case, however, a total AF of 8 was applied to the QCE of 20 mg/kg-day to yield a TRV of 2.5 mg/kg-day. R was set equal to two (versus one) to account for the extrapolation of results between functional groups.

Adult male and female dogs (4 per sex per treatment group) were given gelatin capsules containing aqueous solutions of 2-chlorotoluene at doses of 0, 5, 20, or 80 mg/kg-day for 97 days (Gibson et al. 1974b). Dogs were examined for hematological and biochemical changes. No changes were observed. Hence, a NOAEL of 80 mg/kg-day was established. A total AF of 8 was applied to this NOAEL to yield a TRV of 10 mg/kg-day (IRIS 1994).

D4-3-1.8 2-Hexanone (CAS No. 591-78-6)

In animal studies involving 3-6 months of inhalation exposure to 2-hexanone, both weight loss and decreased rates of weight gain in developing animals were reported. The only lethality data available are from a study by Abdo et al. (1982) where 1 in 5 hens exposed continuously to 200 ppm 2-hexanone died on day 72 of a 90 day study.

An LD_{50} of 2,500 mg/kg was calculated for the gavage administration of 2-hexanone to rats (Smyth et al. 1954). One of 4 hens administered 2-hexanone (70% purity) at 2,000 mg/kg by gavage died (Abou-Donia et al. 1982).

Male rats that were given 2-hexanone at 660 mg/kg/day by gavage in a 90-day study were observed to develop atrophy of the geminal epithelium of the testes (Krasavage et al. 1980).

D4-3-1.9 2-Propanol (CAS No. 67-63-0)

A 1978 study exposed groups of Wistar rats (15/sex/dose) to drinking water containing 0, 50, 100, 200, or 800 ppm (mg/L) allyl alcohol for 15 weeks. Based on water consumption data, these concentration were equivalent to dosages of 0, 4.8, 8.3, 14.0, and 48.2 mg/kg/day for males and 0, 6.2, 6.9, 17.1, and 58.4 for females, respectively. Food intake and growth were depressed at the 100, 200, and 800 ppm dose levels. Results of hematologic and clinical chemistry tests were unremarkable. There were no histopathologic lesions attributable to treatment in any of the organs examined; however, the relative organ weights of the liver, kidney, and spleen were significantly increased in a dose-related fashion at all except the 50 ppm level. Several tests of renal function were performed, indicating impaired renal function in males at greater than or equal to 100 ppm (8.3 mg/kg/day) and in females at greater than or equal to 200 ppm (17.1 mg/kg/day).

The results of the above study are similar to those found by other studies in which rats treated with allyl alcohol in drinking water for 90 days had significantly increased liver and kidney weights at greater than or equal to 250 ppm. Two subchronic inhalation studies were also available in different species in which dose-related increased liver and kidney damage was observed, but the magnitude of these effects are not necessarily comparable to the oral route (IRIS 1994).

D4-3-1.10 2,3,7,8-Tetrachloro dibenzodioxin (CAS No. 51207-31-9)

2,3,7,8-Tetrachloro dibenzodioxin is a confirmed carcinogen with experimental carcinogenic, neoplastigenic, tumorigenic, and teratogenic data. It is one of the most toxic synthetic chemicals. A deadly experimental poison by ingestion, skin contact, and intraperitoneal routes. It is very toxic to some animals, with an LD_{50} of only about 0.6 μg/kg body mass in male guinea pigs. Because of its properties, TCDD is a stable, persistent environmental pollutant and hazardous waste constituent of considerable concern.

Tetrachlorodibenzo-p-dioxin has been shown to be extremely toxic to a number of animal species, the acute oral LD_{50} values ranging from 0.0006 to 0.283 mg/kg, the guinea pig being the most susceptible species. However, it should be emphasized that mortality does not occur immediately, the animals undergoing a slow but progressive decline into a moribund state associated with an increased incidence of infections and the eventual death some 14–28 days after treatment.

Rodents exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) demonstrated severe thymus atrophy. Histologic evaluation of the thymus revealed cortical lymphoid depletion similar to cortisone-induced thymus atrophy. Depressed antibody responses, graft-versus-host, and lympho-proliferative responses were observed at slightly higher doses of TCDD. In addition, increased susceptibility to challenge with the bacteria salmonella bern was noted at low dosages. Depressed antibody responses were also observed in guinea pigs receiving cumulative dosages of TCDD as low as 0.32 μg/kg over an eight-week period. Depressed T-cell function was observed following exposure of adult mice to TCDD, which was associated with an increase in suppresser T-lymphocyte expression and loss of T-lymphocyte cytotoxicity for tumor target cells. Depressed antibody responses and depressed lymphoproliferative responses to mitogens without alteration in cytotoxicity for tumor cells or susceptibility to bacterial or tumor cell challenge in mice exposed to TCDD has been observed by other researchers as well. Decreased antibody plaque responses with no effect on macrophage or NK cell function in TCDD-treated mice have been observed. These results are consistent with an increased susceptibility of TCDD-exposed mice to infection with influenza virus and a lack of effect on a Listeria bacterial challenge. TCDD, and other dioxin isomers, may also suppress serum complement levels in mice, resulting in an increased susceptibility to challenge with Streptococcus pneumoniae infection in these animals.

Exposure to TCDD during thymic organogenesis in rodents has resulted in more severe CMI suppression than that occurring following adult exposure. In some species, in utero exposure via maternal dosing appears to be necessary to induce maximum immunosuppression. At higher dosages, antibody responses and bone marrow stem cell numbers are depressed in most species. Administration of TCDD in utero also results in decreased resistance of offspring to bacterial and tumor cell challenge which correlates with altered CMI in these mice.

Hen pheasants injected with TCDD exhibited delayed-onset body weight and mortality from doses of 6.25, 25, or 100 ug/kg. The lowest dose that produced these responses was 10 ug/kg. At this dose the egg production and hatchibility of the eggs was reduced (Nosek et al. 1992).

D4-3-1.11 2,4-Dimethylphenol (CAS No. 105-67-9)

2,4-Dimethylphenol was administered daily to male and female albino mice by gavage. The animals (30/sex/group) were dosed for 90 days with 5.0, 50.0, or 250 mg 2,4-dimethylphenol/kg/day. Two control groups, untreated and vehicle (corn oil), of similar size were also established. Effects examined included mortality, clinical signs, body weights, food consumption, ophthalmology, hematology and clinical chemistry, organ weights, and gross histopathology. Although 15 deaths occurred during this study (mostly because of errors in technical procedure), only one was considered as possibly treatment-related: a male in the 5 mg/kg/day-dose group died during the first 30 days of the experiment. No significant differences were found between treated and vehicle control groups in mean body weight, body weight gains, food consumption, or eye examinations at any dosage. Toxicologically relevant clinical signs observed only after week 6 in the high-dose groups of both genders included: squinting, lethargy, prostration, and ataxia, with onset shortly after dosing.

At interim sacrifice in female mid- and high-dose groups, blood urea nitrogen (BUN) levels were significantly below vehicle controls; whereas at final sacrifice in the female mid-dose group, BUN levels were significantly higher than vehicle controls. Low-dose males at interim sacrifice had significantly higher cholesterol levels. Significant differences were not found in gross necropsy or histopathological evaluations, or in organ weights, except for an increase in adrenal weights of low-dose females. The LOAEL and NOAEL for this study were 250 and 50 mg/kg/day, respectively (IRIS 1994).

A 14-day gavage study with 2,4-dimethylphenol conducted by the same laboratory that conducted the principal study, revealed lethargy, prostration, and ataxia in males and females in the 250 mg/kg/day-dose group, the same dose at which effects were found in the principal study (U.S. EPA, 1987).

D4-3-1.12 2,4-Dinitrotoluene (CAS No. 121-14-2)

2,4-Dinitrotoluene is the most prevalent dinitrated isomer produced by the nitration of toluene. A carcinogenesis bioassay study by the National Cancer Institute (1978) showed an increased incidence of subcutaneous fibromas in male Fischer 344 rats given 0.02% or 0.008% 2,4-DNT in the feed and increased incidence of mammary gland fibroadenomas in female rats fed higher doses. No carcinogenic effects were observed.

Subchronic and chronic toxicities of 2,4-dinitrotoluene were studied by Ellis et al. (1985) in beagle dogs. The major adverse effects of 2,4-dinitrotoluene in dogs was a neuropathy, characterized by incoordination and paralysis. There were vacuolation, endothelial proliferation, and gliosis of the cerebellums of some affected dogs.

Ellis et al. (1979) tested 2,4-DNT (98% 2,4-DNT and 2% 2,6-DNT) in a chronic oral study using Charles River CD (Sprague-Dawley) rats (38/sex/dose) and CD-1 Swiss mice (58/sex/dose) for 2 years. Rats and mice were fed dietary concentrations of 0, 15, 100, and 700 ppm and 0, 100, 700, and 5,000 ppm, respectively. Mortality was high in all treatment groups; the control group survival rate at 2 years was only 40-45% in rats and 20-30% in mice. In rats the test chemical induced increased incidences of hepatocellular carcinomas in high-dose males (1/25, 2/28, 2/19, 6/30) and a statistically significant increase in the same tumor type in high-dose females (0/23, 0/35, 1/27, 19/35). The incidence of hepatocellular neoplastic nodules was not considered statistically significantly elevated in any of the rat treatment groups. A statistically significant increase in the incidence of benign mammary gland tumors was observed in high-dose female rats (8/23, 9/35, 16/27, 33/35). Most male mice in the high-dose group died before 12 months and were not included in the incidence. In male mice the incidence of kidney tumors (both benign and malignant) was significantly elevated in the mid-dose group (0/20, 4/21, 15/17

for control, low and medium dose groups). No evidence of treatment-related increases in tumor frequency was noted in female mice.

In a 2-year NCI study (1978), 2,4-DNT (greater than 95% purity) was administered in the diet of Fischer 344 rats (50/sex/dose) and B6C3F1 mice (50/sex/dose) at doses of 80 and 200 ppm (rats) and 80 and 400 ppm (mice). Controls consisted of 75 rats/sex and 50 mice/sex. Rats and mice were on test for 78 weeks followed by an additional observation period of 13 to 26 weeks. Only benign tumors were noted. 2,4-DNT induced a statistically significant increase in fibromas of the skin and subcutaneous tissue in male rats (0/71, 7/49, 13/49) and fibroadenomas of the mammary gland in high-dose female rats (13/71, 12/49, 23/50). No statistically significant increase in incidence of tumors was noted in male or female mice.

Leonard et al. (1987) treated groups of 20 F344 male rats with either technical-grade DNT, 2,4-DNT, or 2,6-DNT in the diet for 1 year. There was an untreated control group of 20 rats. Technical DNT (76% 2,4-DNT, 19% 2,6-DNT) (35 mg/kg/day) induced hepatocellular carcinomas in 47% (9/19) of the treated males. 2,6-DNT (99.9% purity) induced hepatocellular carcinomas in 100% (19/19) of the high-dose rats (14 mg/kg/day) and 85% (17/20) of the low-dose (7 mg/kg/day). No tumors were found in controls or rats exposed to 2,4-DNT (99.9 purity) at 27 mg/kg/day. Two low-dose males receiving 2,6-DNT and two males receiving technical DNT developed cholangiocarcinoma (IRIS 1994).

D4-3-1.13 2,4-Dichlorophenoxyacetic acid (CAS No. 94-75-7)

Hematologic, hepatic, and renal toxicity were demonstrated in a study in Fischer rats (strain 344) during subchronic feeding performed at the Hazleton Laboratories (1983). 2,4-D (97.5% pure) was added to the diet chow and fed to the rats for 91 days at doses calculated to be 0.0 (controls), 1.0, 5.0, 15.0, or 45.0 mg/kg/day. In each of the five groups there were 20 animals/sex and 40 animals/treatment group, for a total of 200 animals. Criteria examined to determine toxicity were survival, daily examination for clinical symptomatology, weekly change in body weights, growth rates, food intake, ophthalmologic changes, changes in organ weights, and clinical, gross and histopathologic alterations. The results of the study demonstrated statistically significant reductions in mean hemoglobin (both sexes), mean hematocrit and red blood cell levels (both sexes), and mean reticulocyte levels (males only) at the 5.0 mg/kg/day dose or higher after 7 weeks. There were also statistically significant reductions in liver enzymes LDH, SGOT, SGPT, and alkaline phosphatase at week 14 in animals treated at the 15.0 mg/kg/day or higher doses. Kidney weights (absolute and relative) showed statistically significant increases in all animals at the 15.0 mg/kg/day dose or higher at the end of the experimental protocol. Histopathologic examinations correlated well with kidney organ weight changes showing cortical and subcortical pathology. Increases in ovarian weights, T-4 levels, and a decrease in BUN were reported, but were not considered to be treatment-related.

In a second part of this study, B6C3F1 mice (20/sex/group) were fed the diet chow mixed with 97.5% pure 2,4-D at 0.0, 5.0, 15.0, 45.0 or 90.0 mg/kg bw/day (calculated doses) for 91 days. Criteria to determine toxicity were the same as for rats. The only effect reported at 5 mg/kg/day was increased weight of adrenals in females. Effects at 15 mg/kg/day included altered organ weights and hematologic effects. Kidney weights were not affected below 45 mg/kg/day.

Chronic toxicity and reproduction studies of 2,4-D indicated no adverse effects at dietary levels up to 500 ppm in dogs (approximately 14.5 mg/kg bw/day), up to 1,250 ppm in rats (approximately 62.5 mg/kg bw/day) (Hansen et al. 1971), or at levels of 1,000 ppm in drinking water (50-100 mg/kg bw/day) in pregnant rats (exposed through gestation and for 10 months following parturition) or their offspring (exposed for up to 2 years after weaning) (Bjorklund and Erne, 1966). A secondary reference to

another study reported an increase in mortality among rats whose dams received approximately 50 mg/kg bw/day of 2,4-D in the diet for 3 months before mating and throughout gestation and lactation (IRIS 1994).

D4-3-1.14 4-Chloroaniline (CAS No. 106-47-8)

A study by NCI (National Cancer Institute) in 1979 used groups of 20 and 50 F344 rats of each sex exposed to p-chloroaniline in the diet at concentration of 0, 250 or 500 ppm for 78 weeks followed by an observation period of 24 weeks. Gross and comprehensive histological examinations were performed on all animals after sacrifice. Significantly increased mortality occurred in the high-dose males and decreased average body weight gain occurred in the high-dose females. Nonneoplastic proliferative lesions of the capsule of the spleen (focal fibrosis with subcapsular mesenchymal proliferation) occurred in most of the treated rats. Fibrosis or fatty infiltration of the splenic parenchyma occurred in some of the high-dose males and one of the high-dose females. Splenic lesions did not occur in any of the control rats. This study did not define a NOEL. The 250 ppm level (12.5 mg/kg/day) is considered to be the LOAEL, which when divided by an uncertainty factor of 3,000 results in a RfD of 0.004 mg/kg/day (IRIS 1994).

Information regarding teratogenicity or other reproductive effects of p-chloroaniline is not available.

D4-3-1.15 4-Methylphenol (CAS No. 106-44-5)

In a 90-day subchronic toxicity study by the U.S. EPA in 1986, p-cresol was administered by gavage to 30 Sprague-Dawley rats/sex/dose at 0, 50, 175, or 600 mg/kg/day, once daily. The following parameters were evaluated: body and organ weights, food consumption, mortality, clinical signs of toxicity, and clinical pathology. At sacrifice, animals were necropsied and tissues and organs were subjected to histopathological evaluation. At 600 mg/kg/day of p-cresol, there was a significant reduction in weight gain (15% for females, 25% for males), significantly reduced food consumption at weeks 1 through 7 and 9 in males and significant increased incidence of CNS effects such as lethargy, excessive salivation, tremors, and diarrhea. Also, at 600 mg/kg/day the liver-to-body and kidney-to-body weight ratios were significantly increased, and there was a greater incidence of tracheal epithelial metaplasia compared with the animals in the control, low- or mid-dose groups. At the mid-dose group (175 mg/kg/day) the reduction in weight gain was 5 to 10% in males between weeks 1 and 3; liver-to-body weight ratio was elevated (though not statistically significant) in both sexes, and kidney-to-body weight ratio was significantly elevated in males. Although there was a slight reduction in weight gain and a small increase in kidney-to-body weight ratio at the 50 mg/kg/day level, these effects were not statistically significant.

In a 90-day neurotoxicity study by the U.S. EPA in 1987, 10 Sprague-Dawley rats/sex/dose, were gavaged daily with 0, 50, 75, or 600 mg/kg/day p-cresol. The lowest dose (50 mg/kg/day) caused clinical signs of CNS-stimulation post dosing such a salivation, rapid respiration, and hypoactivity; however, they were low in incidence and sporadic in nature. The highest dose of p-cresol (600 mg/kg/day) produced significant neurological effects, such as increased salivation and urination, tremors, lacrimation, palpebral closure, and rapid respiration. High-dose animals also showed abnormal patterns in the neurobehavioral tests. The NOAEL based on systemic toxicity was 50 mg/kg/day in rats.

In a series of subchronic inhalation studies, rats and guinea pigs exposed to o-cresol at a concentration of 9.0 (plus or minus 0.9) mg/m^3. No effect was seen in guinea pigs. In rats, researchers reported various hematopoietic effects, respiratory tract irritation and sclerosis of lungs.

In a study in 1959, female Sutter mice (27 to 29/group; 2 to 3 months of age) received a single dermal application of 25 uL of 0.3% dimethylbenzanthracene (DMBA) in acetone as the initiator, followed 1 week later by 25 uL of 20% (v/v) o-, m- or p-cresol in benzene twice weekly for 12 weeks. Skin papillomas were evaluated at 12 weeks. Many of the cresol-treated mice died, presumably of cresol toxicity. There was no mortality or evidence of skin papillomas in the benzene control group (benzene weekly after DMBA initiation). The numbers of surviving mice that developed skin papillomas at 12 weeks were as follows: 10/17, o-cresol; 7/14, m-cresol; and 7/20, p-cresol. None of the 12 mice in the benzene control group died or developed skin papillomas.

In another experiment, groups of 20 mice received a single dose (25 uL) of 0.3% DMBA in acetone, followed by twice weekly applications of 5.7% m-cresol in benzene or 5.7% p-cresol in benzene for 20 weeks. No skin papillomas were observed in the 18 surviving benzene control mice; 4/17 m-cresol- and 4/14 p-cresol-treated mice developed skin papillomas. These two experiments indicate that cresols can serve as tumor promoters of a polycyclic aromatic hydrocarbon.

In an acute dermal toxicity study, technical grade o-, m-, and p-cresol caused severe skin damage on at least 2/6 shaved, female, albino New Zealand rabbits within 4 hours of application of 2000 mg/kg of technical grade cresol, 890 mg/kg of o-cresol, 2830 mg/kg of m-cresol, or 300 mg/kg p-cresol (IRIS 1994).

D4-3-1.16 4-Chloro-3-methylphenol (CAS No. 59-50-7)

4-Chloro-3-methylphenol commonly referred to as 4-chloro-m-cresol, is reported to be poisonous by intraperitoneal and subcutaneous routes. It is reported as moderately toxic by ingestion and an allergen. Chlorophenols in general are suspected experimental carcinogens. Most are strong eye and skin irritants and systemic irritants by inhalation, ingestion, and skin contact. They are reported to be mutagenic in general.

In general, chlorinated phenols have been used as wood preservatives that prevent wood rot through their fungicidal action and prevent termite infestation because of their insecticidal properties. Although exposure to these compounds has been correlated with liver malfunction and dermatitis, contaminated polychlorinated dibenzodioxins may have caused some of the observed effects. No other information on the toxicity of 4-Chloro-3-methylphenol was found.

D4-3-1.17 Acenaphthene (CAS No. 83-32-9)

Adult male and female mice (20 each sex/treatment group) were given oral (gavage) doses of acenaphthene for 90 days. Treatment levels were 0, 175, 350, and 700 mg/kg-day. Toxicological evaluations included body weight changes, food consumption changes, mortality, clinical pathology, organ weights, and histopathological evaluations of target organs. No treatment-related effects were observed in any animals in the low dose group. The mid- and high-dose groups experienced cellular hypertrophy in the liver. A TRV of 21.9 mg/kg-day was developed.

D4-3-1.18 Acetone (CAS No. 67-64-1)

Acetone is a common air contaminant that is moderately toxic by various routes. It is a skin and eye irritant and is narcotic in high concentration (Sax and Lewis, 1987).

Acetone was administered via gavage for 90 days to group of albino rats (30 each sex per group) at treatment levels of 0, 100, 500, or 2500 mg/kg-day (EPA 1986b). Body weights, clinical chemistry, hematology, histopathologic parameters, food consumption, and organ weights were measured. No effects were observed at the 100 mg/kg-day dose. Histopathologic studies showed that rats in the 2,500 mg/kg-day group had a marked increase in tubular degeneration of the kidneys and hyaline droplet accumulation with increasing dose.

Inhalation exposure to acetone for a few hours has resulted in rats at concentrations ranging from 16,000 to 50,600 ppm (Bruckner and Peterson 1981) and in guinea pigs from 10,000 to 50,000 ppm (Specht et al. 1939).

No reproductive effects (i.e., no effects on number of implants/litter, percent live pups/litter, percent live pups/litter, or mean percent resorptions/litter were observed in rats or mice in an inhalation developmental study (NTP 1988). No effects were observed on the fertility of male Wistar rats treated with drinking water containing acetone at 1,071 mg/kg/day for 6 weeks (Larsen et al. 1991).

No information on the toxicological effects of acetone avian receptors was located.

D4-3-1.19 Acetonitrile (CAS No. 75-05-8)

Groups of 10 male and 10 female Fischer 344 rats and 10 male and 10 female B6C3F1 mice were exposed to 0, 25, 50, 100, 200, or 400 ppm (0, 42, 84, 168, 336, or 672 mg/m^3) of acetonitrile vapor 6 hours/day for 65 days during a 92-day experimental period (NTP, 1983). The only statistically significant effects observed in rats were decreased mean leukocyte counts in males exposed to greater than or equal to 100 ppm of acetonitrile and females exposed to 400 ppm of acetonitrile. Body weights were slightly increased during the study, with females exposed to greater than or equal to 100 ppm and males exposed to 400 ppm, but there were no statistically significant differences in terminal body weights between test and control rats. Males exposed to 400 ppm of acetonitrile had slightly increased heart-to-body weight ratios, but no cardiac histopathologic alterations. Hepatocyte vacuolization was observed in all groups evaluated, including controls, but the intensity of the vacuolization was slightly greater in the female rats exposed to 400 ppm of acetonitrile.

The results of this study indicated that mice appeared to be more sensitive than rats to similar doses of acetonitrile administered by inhalation. The 100 ppm (168 mg/m^3) dose was a NOAEL in both mice and rats, and the 200 ppm (338 mg/m^3) dose was a LOAEL for hepatic lesions and hematologic changes in mice.

In a 1959 study, researchers exposed three dogs and three rhesus monkeys to 350 ppm (588 mg/m^3) acetonitrile by inhalation for 9 days. Both the monkeys and dogs had pulmonary abnormalities. Brain hemorrhages were observed in all three monkeys, and cloudy swelling of the kidney tubules was noted in two of the three exposed monkeys. The 350 ppm (588 mg/m^3) dose or 41.3 mg/kg/day in monkeys was certainly adverse; monkeys were more sensitive to the toxic effects of acetonitrile than dogs exposed to a similar dose.

The only additional oral toxicity data were provided by teratogenicity studies using hamsters and rats in which researchers treated groups of 6-8 pregnant golden hamsters by gavage with 0, 100, 200, 300, or 400 mg/kg of acetonitrile on gestation day 8. Fetal weight was significantly decreased in all treated groups, fetal resorptions were significantly increased at 200 and 400 mg/kg, and the incidence of malformed offspring was significantly increased at greater than or equal to 300 mg/kg. Signs of maternal toxicity occurred at 400 mg/kg (IRIS 1994).

D4-3-1.20 Acrylonitrile (CAS No. 107-13-1)

Quast et al. (1980a) administered acrylonitrile in drinking water at dose levels of 35, 100, and 300 ppm to 48 Sprague-Dawley rats/sex for 2 years. A statistically significant increase in tumors was observed in the CNS (astrocytomas), Zymbal gland, stomach, tongue, and small intestine for both sexes and in the mammary gland of female rats.

Biodynamics (1980a) administered acrylonitrile in drinking water at doses of 0, 1, and 100 ppm to 100 Sprague-Dawley rats/sex/group. Interim necropsies were performed at 6, 12, and 18 months (10/sex/group). The study was terminated early because of low survival rates. There was increased incidence of astrocytomas of the brain and spinal cord, carcinomas and adenomas of the Zymbal gland or ear canal, and squamous cell carcinomas and papillomas of the forestomach in high-dose animals.

A second study was conducted by Biodynamics (1980b) wherein acrylonitrile was administered in drinking water to 100 Fischer 344 rats/sex/group at dose levels of 1, 3, 10, 30, and 100 ppm and to a control group of 200/sex. Interim necropsies were performed at 6, 12, and 18 months (10/sex/ exposed group and 20/sex/control group). The study was terminated early because of the low survival rate. Increased incidence of tumors (astro- cytomas of the brain and spinal cord, and carcinomas of the Zymbal gland) was seen in dose groups of 3 ppm or higher, and the incidence was dose-dependent. An increased incidence of mammary gland tumors was seen in females at the 100 ppm dose level. In a three-generation reproductive study in rats were exposed to acrylonitrile in drinking water. The second generation showed an increased incidence of cancer (astrocytoma and Zymbal gland) at the 500 ppm exposure level (Beliles, 1980).

Maltoni et al. (1977) administered acrylonitrile in olive oil 3 times/week for 52 weeks to Sprague-Dawley rats in doses of 0 ppm to 75 rats/sex and 5 ppm to 40 rats/sex. Increased incidence of tumors of the mammary gland and forestomach was observed in female rats. In another study (Biodynamics, 1980c), acrylonitrile was administered at doses of 0, 0.10, and 10.0 mg/kg/day for 5 days to 70 Sprague-Dawley rats/sex/group. The study was terminated at 20 months. Statistically significant increased incidences of brain (astrocytoma) and Zymbal gland tumors were observed in the high-dose group. A statistically significant increased incidence of stomach and intestinal tumors was observed in males and of the mammary gland in females.

In a second study by Quast et al. (1980b), acrylonitrile was administered by inhalation at 0, 20, and 80 ppm to 100 male and female Sprague-Dawley rats for 6 hours/day, 5 days/week for 2 years. A statistically significant increase was observed in tumors of the CNS and other sites.

Acrylonitrile was also administered by inhalation at lower doses of 0, 5, 10, 20, and 40 ppm, 4 hours/day, 5 days/week for 12 months to 30 Sprague-Dawley rats/sex/group by Maltoni et al. (1977). This resulted in a statistically significant increase of mammary tumors in males and skin carcinomas in females (IRIS 1994).

D4-3-1.21 Aluminum (CAS No. 7429-90-5)

Aluminum is the third most abundant element in the earth's crust, exceeded only by oxygen and silicon. However, it is found only in trace amounts in biological organisms. Aluminum has not been proven essential to animals (Schroeder and Nason, 1971), but indirect evidence suggests it may be (Sorenson et al. 1974). Its concentration in tissues changes in a circadian rhythm and with other changes

in biological activity (Sorenson et al. 1974). If a biological requirement for aluminum exists, it has not been quantified.

There are no reports in the technical literature of mammal toxicity attributed to naturally occurring aluminum in the environment. It seems unlikely that grazing animals could achieve toxic concentrations naturally (Gough et al. 1979). An acute toxic dietary concentration of 20,000 mg/kg for the rat was reported by Gough et al. (1979). NAS/NAE (1972) reported that a level of 4,000 mg/kg of aluminum in the diet of chicks caused phosphorus deficiency. EPA (1983) reports a phytotoxic concentration for vegetation from soluble aluminum of 2 to 14 mg/kg.

Aluminum ingested by mammals is readily eliminated by the kidneys and does not bioaccumulate or biomagnify. Studies with laboratory rats indicated that tissue concentrations of aluminum between treated and control animals were similar 7 days after withdrawal of dietary aluminum. An acute toxic dietary concentration of 20,000 mg/kg for the rat was reported by Gough et al. (1979). NAS/NAE (1972) reported that a level of 4,000 mg/kg of aluminum in the diet of chicks caused phosphorus deficiency leading to rickets, weakness, and possibly death. There is no indication from several studies that aluminum alters the reproductive capabilities of rats or mice (Clement Associated, Inc., 1990). McKee and Wolf (1963) report that aluminum is not highly toxic to wildlife.

It is unlikely that aluminum poses a toxicological risk to wildlife. Even though concentrations of aluminum that exceeded guidelines were reported from sediment, soil, and subsurface water media, the scientific literature indicates that wildlife species can probably bioaccumulate and regulate large concentrations of this metal without harm.

D4-3-1.22 Aluminum nitrate nonahydrate [Al $(NO_3)_3.9H_2O$] (CAS No. 7784-27-2)—(See also Nitrate)

Aluminum compounds are generally not toxic except in cases of high experimental doses or prolonged inhalation (Venugopal and Luckey 1978). The lack of toxic effect is partly attributable to the poor absorption of aluminum by the gut and lungs. Most large oral doses of aluminum are excreted if the kidneys are intact. In mammals, the mechanism of aluminum toxicity appears to be the impairment of phosphorous metabolism. The neurotoxicity of aluminum is due to its binding with nervous tissue, which can cause massive neurofibrillary degeneration. Chronic administration of aluminum (as aluminum chloride) caused impaired growth in second and third generation animals without affecting litter size. The normal concentration of aluminum in the mammalian brain is 1 to 2 kg/g. Rats are the least sensitive species to aluminum toxicosis, while cats and rabbits are the most sensitive species.

A TDL of 1,620 mg/kg-day was available for rats (omnivores) exposed orally to aluminum nitrate nonahydrate (Teratology 1988). In an additional study, a NOAEL of 6.0 mg/kg-day was established when 3- to 4- week old rats fed 6 to 10 mg/kg aluminum hydroxide exhibited significantly retarded growth (Thurston et al. 1972). The Thurston et al. (1972) study was used to develop a TRV for omnivores.

NOAELs of 1,900 and 1,200 mg/kg-day were reported by rabbits (Nekipelov 1966), sheep (Thompson et al. 1959), and cattle (Valdivia et al. 1978) exposed to $Al(NO_3)_2$, $AlSO_4$, and $AlC1_2$, respectively. A TRV was developed for herbivores and carnivores using the Nekipelov (1966) data.

For avian receptors, a NOAEL of 3,300 mg/kg-day (Miller and Kifer 1970) and a NOAEL of 324 mg/kg-day (Cakir et al. 1978) were available for chicken and turkeys, both of which belong to the order galliformes and are omnivores. The study identifying a subchronic NOAEL for 1-day old turkeys

exposed to $Al(SO_4).18H_2O$ (Cakir et al. 1978) was selected for TRV development because it was the most conservative of the values and a sensitive life stage was examined.

D4-3-1.23 Anthracene (CAS No. 120-12-7)

Anthracene was administered to groups of 20 male and female CD-1 (ICR)BR mice by oral gavage at doses of 0, 250, 500, and 1,000 mg/kg/day for at least 90 days. Mortality, clinical signs, body weights, food consumption, ophthalmology findings, hematology and clinical chemistry results, organ weights, organ-to-body weight ratios, gross pathology, and histopathology findings were evaluated. No treatment-related effects were noted. The no-observed-effect level (NOEL) is the highest dose tested (1,000 mg/kg/day).

In a chronic bioassay (Schmahl, 1955), a group of 28 BD I and BD III rats received anthracene in the diet, starting when the rats were approximately 100 days old. The daily dosage was 5 to 15 mg/rat, and the experiment was terminated when a total dose of 4.5 g/rat was achieved, on the 550th experimental day. The rats were observed until they died, with some living more than 1,000 days. No treatment-related effects on life span or gross and histological appearance of tissues were observed. Body weights were not mentioned, and hematological parameters were not measured (IRIS 1994).

A single dermal application of 10 um anthracene (purity not stated) in benzene was administered to 30 female CD-1 mice; this initial application was followed 7 days later by twice-weekly applications of 5 um 12-0-tetradecanoyl phorbol-13-acetate (TPA) for 35 weeks. Survival in the group was 93% after 35 weeks. By week 20 of the test, 2/28 mice had developed skin tumors; this increased to 4/28 by week 35. In the control group, in which 30 mice received only the TPA applications, a mouse developed a skin tumor at week 25 (Scribner, 1973).

D4-3-1.24 Antimony (CAS No. 7440-36-0)

Antimony causes a number of toxic effects in animals, including suppression of weight gain, shortened life span, and damage to liver, heart, thyroid, and kidneys. Trivalent compounds (e.g., antimony trioxide, antimony trisulfide) are about 10 times more toxic than pentavalent forms. The gastrointestinal absorption of trivalent antimony is about 15 - 36% (Weitz and Ober 1965; van Bruwaene et al. 1982; Gerber et al. 1982). The acute toxicity of antimony trioxide is low, with an oral LD_{50} in rats of greater than 20 g/kg (Smyth and Carpenter 1948).

In chronic studies, 5 mg/L potassium antimony tartrate (approximately 0.35 mg/kg-day) in drinking water is associated with slightly decreased life spans in rats (Schroeder et al. 1970) and female mice (Schroeder et al. 1968; Kanisawa and Schroeder 1969). Endpoints examined in these chronic (lifetime) studies included growth and body weight, median life span, longevity, tumor incidence, and histopathology. Other ecologically relevant endpoints (e.g., reproduction) were not examined, and only one dose was administered. Although rats appeared to be more sensitive than mice in these studies, the effects reported are of questionable ecological significance.

No information on the toxicological effects of antimony on avian receptors was located.

D4-3-1.25 Arsenic (CAS No. 7440-38-2)

Arsenic is a metalloid element that is widespread in all environmental media, making up about 0.0005% of the earth's crust. Arsenic is commonly present in living organisms and is constantly being oxidized, reduced, or metabolized.

The potential toxicity of arsenic to any organism is dependent on its chemical form. Inorganic arsenicals are generally more toxic than organic arsenicals, and trivalent forms are more toxic than pentavalent forms. Toxicity is related to aqueous solubility, and the order of toxicity (from greatest to least) is arsines > inorganic arsenites > organic trivalent compounds > inorganic arsenates > organic pentavalent compounds > arsonium compounds > elemental arsenic (Eisler 1988a).

Chemical properties contributing to arsenic's toxicity include its ability to bind to protein sulfhydryl groups and to substitute for phosphorus in some biochemical reactions. These chemical properties may also be responsible for arsenic's apparent essentially in several mammalian species (Frost 1983; Uthus 1992). In fact, arsenical feed additives promote growth in a number of agricultural species (Eisler 1988a). Recent studies have suggested that arsenic has a physiological role in the formation of various metabolites of methionine metabolism (Uthus 1992). The arsenic requirement for growing chicks and rats is approximately 25 mg/kg diet (Uthus 1992). Species differences in the pharmacokinetic disposition of arsenic have significant effects on their sensitivity to its toxic effects. In addition, animals exposed to sublethal levels of arsenic can develop tolerance to subsequent exposures (Eisler 1988a).

A subacute study using domestic sheep was documented (Eisler 1988a) in which an NOEL endpoint using 2.3 mg/kg-day was reported. An LOAEL of 1.5 mg/kg-day was reported in a chronic study using sodium arsenate in rats (Byron et al. 1967). The data did not show a good dose-response curve in the low dose range.

The National Academy of Sciences reported a LD_{50} of 39 mg/kg-day using sodium arsenite in mallards.

D4-3-1.26 Asbestos (CAS No. 1332-21-4)

Asbestos is a mineral fiber composed of hydrated mineral chain silicates. Because asbestos has an unusual combination of properties, such as its fibrous nature, its fire resistance, its inertness, and its insulation properties, it has found many uses. It has been thought to induce lung cancer in human workers that have been exposed to asbestos over the course of their lifetime.

A study by Smith et al. (1965) using 45 hamsters fed 1% asbestos over their lifetime revealed no tumors or lesions in their lungs. A second experiment by Donham et al. (1979) on rats given 5.9, 17.1, or 29.4 mg of chrysotile by gavage showed gross lesions observed in the colon tissues. Several gross tumors were observed.

Gross et al. (1967) exposed 61 white male rats (strain not reported) to 86 mg chrysotile asbestos dust/m^3 for 30 hours/week for 16 months. Of the 41 animals that survived the exposure period, 10 had lung cancer. No lung cancer was observed in 25 controls.

Reeves (1976) exposed 60 to 77 rats/group for 4 hours/day, 4 days/week for 2 years to doses of 48.7 to 50.2 mg/m^3 crocidolite, 48.2 to 48.6 mg/m^3 amosite and 47.4 to 47.9 mg/m^3 chrysotile. A

5 to 14% incidence of lung cancer was observed among concentration groups and was concentration-dependent.

Wagner et al. (1974) exposed CD Wistar rats (19 to 52/group) to 9.7 to 14.7 mg/m^3 of several types of asbestos for 1 day to 24 months for 7 hours/day, 5 days/week. A duration-dependent increased incidence of lung carcinomas and mesotheliomas was seen for all types of asbestos after 3 months of exposure compared with controls.

F344 rats (88 to 250/group) were exposed to intermediate range chrysotile asbestos (1291E+8 f/g) in drinking water by gavage to dams during lactation and then in diet throughout their lifetime (NTP, 1985). A statistically significant increase in incidence of benign epithelial neoplasms (adenomatous polyps in the large intestine) was observed in male rats compared with pooled controls of all NTP oral lifetime studies (3/524). In the same study, rats exposed to short range chrysotile asbestos (6081E+9 f/g) showed no significant increase in tumor incidence.

Ward et al. (1980) administered 10 mg UICC amosite asbestos 3 times/week for 10 weeks by gavage to 50 male F344 rats. The animals were observed for an additional 78 to 79 weeks post-treatment. A total of 17 colon carcinomas were observed. This result was statistically significant compared with historical controls; no concurrent controls were maintained.

Syrian golden hamsters (126 to 253/group) were exposed to short and intermediate range chrysotile asbestos at a concentration of 1% in the diet for the lifetime of the animals (NTP, 1983). An increased incidence of neoplasia of the adrenal cortex was observed in both males and females exposed to intermediate range fibers and in males exposed to short range fibers. This increase was statistically significant by comparison to pooled controls but not by comparison to concurrent controls. NTP suggested that the biologic importance of adrenal tumors in the absence of target organ (GI tract) neoplasia was questionable (IRIS 1994).

D4-3-1.27 Barium (CAS No. 513-77-9)

The symptoms of acute barium poisoning in humans and other mammals are excessive salivation, vomiting, colic, violent diarrhea, tremors, muscular paralysis, and paralysis of the central nervous system. Strong vasoconstriction, due to the direct barium stimulation of arterial muscles, raises the blood pressure; high blood pressure causes hemorrhage in the stomach, intestines, and kidneys. Symptoms of chronic barium poisoning are similar but of lesser severity. Guinea pigs, dogs, and mice are more susceptible to barium toxicity than are chickens, rats, and rabbits; humans are also susceptible to barium toxicity. The toxicity of soluble barium salts is higher than that of corresponding salts of calcium, magnesium, and strontium (Venugopal and Luckey, 1978).

Barium's acute toxicity is low, with LD_{50}s in experimental animals consistently greater than 100 mg/kg (ATSDR 1992a). High barium concentrations (2 to 10 ppm) in human drinking water have been reported to be associated with elevated cardiovascular mortality, hypertension, and other cardiovascular effects (ATSDR 1992a).

Results in animal studies indicate that acute, intermediate, and chronic oral exposure to barium is not associated with any adverse hematological effects. Developmental effects reported in a study by Tarasenko et al. (1977) in rats reported effects in offspring included increased mortality, increased leukocyte count, disturbances in liver function, and increased urinary excretion of hippuric acid.

Increased blood pressure, depressed cardiac contractility and conduction, and lower cardiac ATP content were observed in rats chronically exposed to 10 to 100 mg Ba/L in drinking water (Perry et al. 1983, 1985, 1989; Kopp et al. 1985). The NOAEL exposure level identified in these studies was 1 mg/L, or approximately 0.5 mg/kg/day.

No information on the toxicological effects of barium on avian receptors was located.

D4-3-1.28 Benzene (CAS No. 71-43-2)

Benzene has been shown to be carcinogenic in animals. Benzene also causes CNS depression, narcosis, and death in various animal species. Leukopenia (A reduction in white blood cells) is the most commonly reported effect in laboratory animals chronically exposed to benzene.

Adult male and female rats and mice (50 per sex per treatment group) were given oral doses of benzene for 103 weeks. Dose levels were 0, 50, 100, or 200 mg/kg-day for rats and 0, 50, 250, or 500 mg/kg-day for mice (NTP, 1986). All animals showed increased incidence of carcinomas of the Zymbal gland. Male and female rats had oral cavity tumors and males showed an increased incidence of skin tumors. Mice of both sexes had increased incidence of lymphomas and lung tumors. In general, the increased incidence observed was dose-related.

Nawrot (1979) reported that mice were administered benzene by gavage at three dose levels. At the highest two doses (0.5 and 1.0 mL/kg/d), significantly increased maternal mortality and embryonic resorption resulted. Fetal weights were significantly reduced at all three dose levels.

D4-3-1.29 Benzine (CAS No. 8032-32-4)

An LD_{50} of 40 mg/kg-day was reported for rats (omnivores) exposed intravenously to benzine (Pharm. Chem. J., 1973, TOXNET).

No data on the toxicological effects of benzine to avian receptors were available. No other information for the toxicity of benzine was found.

D4-3-1.30 Benzo(a)anthracene (BA) (CAS No. 56-55-3)

BA produced tumors in mice exposed via gavage, intraperitoneal injection, subcutaneous injection, and topical application.

Benz[a]anthracene administration caused an increase in the incidence of tumors by gavage (Klein, 1963); dermal application (IARC, 1973); and both subcutaneous injection (Steiner and Faulk, 1951; Steiner and Edgecomb, 1952) and intraperitoneal injection (Wislocki et al. 1986) assays. A group of male B6AF1/J mice was exposed to gavage solutions containing 3% benz[a]anthracene in Methocel-Aerosol O.T. (dioctyl ester of sodium sulfo-succinic acid), 3 doses/week for 5 weeks (total dose of approximately 225 mg/mouse, 500 mg/kg/day) or the vehicle (Klein, 1963). Mice were evaluated for tumors on days 437-444 and 547 after treatment was initiated. A statistical analysis was not reported. Increased incidences of pulmonary adenoma and hepatoma in treated vs. control mice were reported by the authors at both observation times. The incidence of pulmonary adenoma at 437 to 444 days was 37/39 (95%) in treated animals vs. 10/38 (26%) in controls; whereas at 547 days, 19/20 (95%) treated animals and 7/20 (35%) controls had pulmonary adenomas. The incidence of hepatomas at 437 to 440 days was 18/39

(46%) in treated animals compared with 0/38 among the vehicle controls. After 547 days, the hepatoma incidences increased to 20/20 for the treated animals versus 2/20 (10%) for vehicle controls.

Mice (strain and sex not specified) were exposed to a single gavage dose of 0.5 mg benz[a]anthracene in mineral oil (approximately 17 mg/kg). No tumors were reported in 13 mice examined 16 months after exposure. In another part of the study, multiple gavage treatments, 8 or 16 treatments at 3-7 day intervals over a 16-month period, resulted in forestomach papillomas in 2/27 treated mice compared with 0/16 in vehicle controls (Bock and King, 1959).

Groups of male and female CD-1 mice (n=90 to 100) received intraperitoneal injections of benz[a]anthracene in DMSO on days 1, 8, and 15 of age (total dose = 638 ug/mouse) (Wislocki et al. 1986). Tumors were evaluated in animals that died spontaneously after weaning and in all remaining animals at 1 year after exposure. In treated male mice, a statistically significant increase in the incidence of liver adenomas or carcinomas (31/39 treated vs. 2/28 controls) occurred; 25/39 had carcinomas. Female mice did not develop liver tumors. The incidence of pulmonary adenomas or carcinomas in benz[a]anthracene-treated males (6/39, with a majority of adenomas) was increased but not statistically significantly relative to the vehicle controls (1/28). In the female mice, however, the incidence of pulmonary adenomas was significantly elevated in the treated group (6/32) when compared with vehicle controls (0/31) (IRIS 1994).

D4-3-1.31 Benzo(a)pyrene (BaP) (CAS No. 50-32-8)

BaP caused cancer in four rodent species and several primates when exposed via gavage, dietary, inhalation, intratracheal instillation, dermally, and subcutaneous injection. Repeated BaP administration has been associated with increased incidence of total tumors and tumors at the site of exposure. Distant site tumors have also been observed by various routes of administration. BaP administered via gavage to mice, rates, and hamsters has caused an increased incidence in stomach tumors.

Brune et al. (1981) fed BaP (reported to be highly pure) to rats 5 times a week until dead or moribund. This treatment resulted in an average dose of 0.11 mg/kg-day. There were 32 rats per sex in both the treatment and control group. Histological examinations were performed on each rat. The combined incidence of forestomach, larynx, and esophagus tumors was 10 out of 64 in the test group and 3 out of 64 in the control group. A trend analysis showed a statically significant tendency for the proportion of animals with tumors to increase steadily with dose (IRIS).

The animal data consist of dietary, gavage, inhalation, intratracheal instillation, dermal and subcutaneous studies in numerous strains of at least four species of rodents and several primates. Repeated BAP administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. Distant site tumors have also been observed after BAP administration by various routes. BAP is frequently used as a positive control in carcinogenicity bioassays.

Intraperitoneal BAP injections have caused increases in the number of injection site tumors in mice and rats (reviewed in U.S. EPA, 1991a). Subcutaneous BAP injections have caused increases in the number of injection site tumors in mice, rats, guinea pigs, hamsters and some primates (IARC, 1983; U.S. EPA, 1991a). BAP is commonly used as a positive control in many dermal application bioassays and has been shown to cause skin tumors in mice, rats, rabbits and guinea pigs. BAP is both an initiator and a complete carcinogen in mouse skin (IARC, 1983). Increased incidences of distant site tumors have also been reported in animals as a consequence of dermal BAP exposure (reviewed in U.S. EPA, 1991a).

BAP has also been reported to be carcinogenic in animals when administered by the following routes: iv.; transplacentally; implantation in the stomach wall, lung, renal parenchyma and brain; injection into the renal pelvis; and vaginal painting (U.S. EPA, 1991a).

D4-3-1.32 Benzo(b)fluoranthene [B(b)Fl] (CAS No. 205-99-2)

In a lifetime implant study, 3-month-old female Osborne—Mendel rats (35/group) received a single lung implant of either 0.1 mg (0.4 mg/kg), 0.3 mg (1.2 mg/kg) or 1 mg (4.1 mg/kg) benzo[b]fluoranthene in 0.05 mL of a 1:1 (v:v) mixture of beeswax and trioctanoin (Deutsch-Wenzel et al. 1983). Controls consisted of an untreated group and a group receiving an implant of the vehicle. The median survival times were: 118, 104, 110, 113, and 112 weeks, for the untreated, vehicle control, low-, mid- and high-dose groups, respectively. The incidences of epidermoid carcinomas and pleomorphic sarcomas in the lung and thorax (combined) were: untreated controls, 0/35; vehicle controls, 0/35; low-dose group, 1/35; mid-dose group, 3/35; and high-dose group, 13/35. These incidences showed a statistically significant dose-response relationship.

Groups of 15 to 17 male and 17 to 18 female CD-1 mice received i.p. injections of benzo[b]fluoranthene in DMSO on days 1, 8, and 15 after birth (total dose was approximately 126 ug/mouse) and were sacrificed at 52 weeks of age (LaVoie et al. 1987). A statistically significant increase in the incidence of liver adenomas and hepatomas (combined) occurred in treated males (8/15) relative to vehicle controls (1/17), but not in females. Lung adenomas (2/15 males, 3/17 females) were reported in treated animals, whereas none were found in controls (IRIS 1994).

D4-3-1.33 Beryllium (CAS No. 7440-41-7)

Beryllium is a metal with a complicated coordination chemistry. It can form complexes, oxycarboxylates, and chelates with a variety of materials. Although little information concerning adsorption of beryllium is available, based on its geochemical similarity to aluminum, it is expected to be adsorbed onto clay mineral surfaces at low pH levels and to be complexed into some insoluble compounds at high pH. In most natural environments, beryllium is likely to be present in sorbed or precipitated, rather than dissolved, form (EPA 1985).

Limited toxicity data is available for oral exposure to beryllium. Decreases in growth have been reported for rats exposed to beryllium. However, significant changes in organs and other physicochemical changes were not reported. Data on the teratogenicity or reproductive effects of beryllium are limited.

No adverse effects were observed in a rat chronic oral study at 5 mg/L beryllium in drinking water (Schroeder and Mitchener 1975). This study was used as the basis for deriving a TRV of 3.57 mg/kg/day.

Beryllium has been reported to produce embryolethality and teratogenicity in chick embryos (Puzanova et al. 1978) when administered subgerminally. However, no dietary toxicity studies from beryllium exposure to birds was located, and a TRV was not derived.

D4-3-1.34 Boron (CAS No. 7440-42-8)

Boron is a poison by ingestion and a moderate fire hazard in the form of dust when exposed to air or by chemical reaction. It is an explosion hazard in the form of dust which ignites on contact with air. The routes of entry are inhalation of dust, fumes, aerosols, and ingestion. Boron compounds may irritate

the nasal mucous membranes, the respiratory tract, and eyes. Acute poisoning in man from boric acid or borax is usually the result of application of dressings, powders, or ointment to large areas of burned or abraded skin, or accidental ingestion (Sittig 1981).

Studies reported in ATSDR (1991) and Weir and Fisher (1972) were used to develop TRVs for boron. Male and female rats were given boron in feed during a 2 year, 3 generation study during which a NOAEL of 17.5 mg/kg-day was established. A subchronic (38-week) study in which dogs were fed boron established a NOAEL of 8.75 mg/kg-day.

No data was located on the toxicological effects of boron on avian or reptilian receptors was located.

D4-3-1.35 Butyl alcohol (n-Butanol) (CAS No. 71-36-3)

N-butanol is a poison by ingestion and is moderately toxic via the dermal route and moderately irritating via the inhalation route to humans. The use of normal butyl alcohol is reported to have resulted in irritation of the eyes, with corneal inflamation, slight headache and dizziness, slight irritation of the nose and throat, and dermatitis. Animal experiments have shown the butyl alcohols to possess toxic properties (Sax and Lewis, 1987).

Four groups of male and female rats (30/sex/group) were dosed daily by gavage with 0, 30, 125, and 500 mg/kg-day of butanol for 13 weeks (EPA, 1986). Ataxia and hypoactivitiy were consistently observed in high-dosed (500 mg/kg-day) males and females during the final 6 weeks of the dosing period. Thus, the 125 mg/kg-day dose of butanol is considered a NOAEL for central nervous system effects in rats (IRIS 1994).

No information on the toxicological effects of n-butanol on avian receptors was located.

D4-3-1.36 Butylbenzylphthalate (BBP) (CAS No. 85-68-7)

Limited data on the carcinogenic potential of BBP in animals are available. Female rats fed 0, 6,000, and 12,000 mg/kg daily for 103 weeks exhibited a significant increase in mononuclear cell leukemia or lymphoma at the high dose rate. The qualitative weakness of the carcinogenic response does not provide a compelling basis to model dose-response data. Subchronic exposure to BBP by rats caused hematological effects, kidney atrophy, and testicular lesions at some of the higher dose levels. Male rats given BBP by gastric intubation for 14 days experienced liver morphological and biochemical changes at a dose rate of 160 mg/kg-day.

The National Toxicology Program (1985) conducted a 26-week subchronic study of potential toxic effects of BBP. Fifteen male rats per group were fed either 0, 17, 51, 159, 470, or 1,417 mg/kg-day in the diet. There were no deaths attributable to BBP toxicity. Terminal mean organ weight was significantly lower in the 1,417 mg/kg-day group for the heart, kidney, lung, seminal vesicles, and testes. Animals in this group also exhibited significantly increased absolute liver weights and increased liver-to-body weight ratios. No adverse effects were observed at the 159 mg/kg-day dose level or below. The NOAEL of 159 mg/kg-day was used to derive the TRV for BBP.

The only other information on subchronic effects is reported by Krauskopf (1973) from an unpublished study by Monsanto (1972). Rats fed diets containing 0.25% (125 mg/kg/day) and 0.5% (250 mg/kg/day) for 90 days showed no toxic effects. Liver weights were increased in animals fed diets

containing 1.0, 1.5, or 2.0% (500, 750, or 1,000 mg/kg/day, respectively) for 90 days, and a mild decrease in growth rate was reported for the 1.5 and 2.0% groups. No other hematologic, histopathologic or urinalysis effects were observed. When dogs were administered gelatin capsules containing doses equivalent to 1.0, 2.0, or 5.0% of the daily diet (10,000 20,000 and 50,000 ppm) for 90 days, no effect on hematological parameters, urinalysis or liver and kidney functions were observed. A bioassay was performed by the NTP (1982) to evaluate the carcinogenic potential of orally administered butyl benzyl phthalate (BBP) to both rats and mice. Dietary levels of 0, 6,000, and 12,000 ppm BBP were fed to groups of 50 male and 50 female F344 rats and 50 male and 50 female B6C3F1 mice for 103 weeks. The male rats at both dose levels experienced high mortality within the first 30 weeks of the study due to apparent internal hemorrhaging; all male rats were, thus, terminated at 30 weeks. No chronic toxicity or carcinogenic effects were observed in male or female mice (IRIS 1994).

No information on the toxicological effects of BBP on avian receptors was located.

D4-3-1.37 Cadmium (CAS No. 7440-43-9)

Cadmium is found naturally in the environment from chemical weathering of rocks. It is generally found in soil as free cadmium compounds (ATSDR 1993). No evidence suggests cadmium is biologically essential (Eisler 1985a). Cadmium is not reduced or methylated by microorganisms (ATSDR 1993).

Birds and mammals are comparatively resistant to cadmium toxicity as compared to aquatic species. Sublethal effects of cadmium include growth retardation, anemia, and testicular damage (Hammons et al. 1978) as cited in Eisler (1985a). Cadmium readily reacts with sulfhydral groups and may inhibit enzymatic reactions (Eisler 1985a). Bioaccumulation of cadmium has been reported in aquatic systems, however, only lower trophic levels exhibit biomagnification (Eisler 1985a). Accumulation of cadmium in avian species has been reported in liver and kidneys.

Chickens exposed to cadmium in the diet had reduced growth rates in a study by Pritzl et al. (1974). Behavioral changes were observed in young American black ducks when parents were fed 4 ppm cadmium for 4 months before egg-laying (Heinz and Haseltine 1983, as cited in Eisler, 1985a).

D4-3-1.38 Carbon disulfide (CAS No. 75-15-0)

Carbon disulfide affects the central nervous system, cardiovascular system, eyes, kidneys, liver, and skin. It may be absorbed through the skin as a vapor or liquid, inhaled, or ingested.

A NCTR-NTP oral study of 1984 observed 25 mg/kg/day in rabbits as an FEL (fetal resorption). Fetotoxicity band fetal malformations in this study were not observed in rats at the lowest level (100 mg/kg/day) of CS2 exposure. The data from this study suggest that the rabbit fetus is more sensitive than the rat fetus to CS2-induced toxicity.

Hardin et al. (1981) observed no effects on fetal development in rats or rabbits following inhalation exposure to 62.3 or 124.6 mg/m^3, which corresponds to estimated equivalent oral dosages of 5 and 10 mg/kg for rats, and 11 and 22 mg/kg for rabbits. The highest NOEL from this study, 22 mg/kg for the rabbit, should not be used for an RfD estimate because adverse effects were seen in rabbit fetuses following oral exposure of pregnant does to 25 mg/kg (Jones-Price et al. 1984a,b). Therefore, the highest NOAEL that is below an effect level is the estimated low dose from the Hardin et al. (1981) inhalation study using rabbits.

D4-3-1.39 Carbon tetrachloride (CAS No. 56-23-5)

Carbon tetrachloride has caused liver tumors in rats, mice, and hamsters exposed via several different routes. In animals, the primary effect associated with short-term exposure to carbon tetrachloride is liver damage (Proctor and Hughes, 1978). Oral doses as low as 15 mg/kg altered liver biochemistry (Adams et al. 1952), while high doses resulted in death within hours from CNS depression (IARC, 1979). Rats given 47 to 160 mg/kg-day carbon tetrachloride via corn oil gavage for 78 weeks developed hepatocellular carcinomas and neoplastic liver nodules.

Male rats were given 1, 10 or 33 mg/kg carbon tetrachloride by corn oil gavage. Liver lesions (mild centrilobular vacuolization and significant increases in serum sorbitol dehydrogenase activity) were observed in the 10 and 33 mg/kg groups in a dose related manner (Bruckner et al. 1986). The critical dose was adjusted for treatment schedule to yield a dose of 7.1 mg/kg-day.

Fifty Osborne—Mendel rats/sex were administered carbon tetrachloride by corn oil gavage at 47 and 94 mg/kg/injection for males and 80 and 159 mg/kg for females 5 times/week for 78 weeks. At 110 weeks, only 7/50 high-dose males and 14/50 high-dose females survived; 14/50 low-dose males and 20/50 low-dose females survived. The incidence of hepatocellular carcinomas was increased in animals exposed to carbon tetrachloride as compared with pooled colony controls. The apparent decrease in the incidence of hepatocellular carcinomas in high-dose female rats compared with the low-dose females (1/14 vs. 4/20, respectively) was attributed by the authors to increased lethality before tumors could be expressed (NCI, 1976a,b, 1977).

No information on the toxicological effects of carbon tetrachloride avian receptors was located.

D4-3-1.40 Cerium Chloride (CAS No. 7790-86-5)

Gastrointestinal absorption of cerium is poor for most mammals. The poor absorption of cerium by mammals led to its use as a nutritional marker. Insoluble compounds, such as cerium oxides, are generally non-toxic when ingested.

Intravenous injection of 3 mg Ce/kg (as $CeCl_3$) caused liver damage in rats. Cerium salts, such as $CeCl_3$, appear to affect the adrenal glands (Venugopal and Luckey, 1978). An oral LD_{50} of 2,111 mg/kg-day is reported for rats (Environ. Qual. Safety Suppl., 1:1, 1975; RTECHS). An oral LD_{50} of 5,277 mg/kg-day is reported for mice (Environ. Qual. Safety Suppl., 1:1, 1975; RTECHS). An oral LD_{50} of 211 mg/kg-day is reported for frogs (Environ. Qual. Safety Suppl., 1:1, 1975; RTECHS). No other information for the toxicity of cerium chloride was found.

D4-3-1.41 Chloroform (CAS No. 67-66-3)

Chloroform is a central nervous system depressant, and chronic exposure may cause liver and kidney damage (Proctor and Hughes, 1978). Liver effects are more characteristic of chronic exposures than kidney injury or central nervous system depression (Sittig, 1981). Sex-linked differences in effects associated with exposure to chloroform have been observed. Females tend to experience more severe effects, probably due to chloroform's affinity for adipose tissue (Fry et al. 1972). Chloroform is considered highly fetotoxic but not teratogenic (Schwetz et al. 1974). Mice given 60 mg/kg-day chloroform via toothpaste gavage for 1.5 years exhibited an increased incidence of renal cortex tumors (Roe et al. 715).

Thompson et al. (1974) evaluated the potential for fetotoxic effects by administering 20, 35, 50, or 126 mg/kg-day chloroform to rats and rabbits via gavage during gestation. No adverse maternal or fetal effects were observed. A study conducted by Ruddick (1980) supports these findings. Ruddick (1980) gave rats 100 mg/kg-day via gavage during gestation and observed no adverse effects.

Male and female beagle dogs (12 each group) were given 15 or 30 mg/kg chloroform in gelatin capsules for 7.5 years (Heywood et al. 1979). The minimum dose adjusted for treatment schedule is 12.9 mg/kg-day. A dose-related increase in SOGT levels and nodules of altered hepatocytes were observed at both treatment levels. This critical study was of chronic duration, used a fairly large number of dogs, and measured multiple endpoints but only two treatment doses were used and a NOEL was not established (IRIS 1994).

No information on the toxicological effects of carbon tetrachloride avian receptors was located.

D4-3-1.42 Chromium(III) (CAS No. 7440-47-3)

Chromium(III) is an essential nutrient (required for insulin function) in mammals. However, it is interconvertible in the environment with the more toxic species chromium(VI), depending primarily on the redox potential and pH of soil (Bartlett 1991).

Approximately 0.5 to 3% of ingested inorganic chromium((III) compounds are absorbed by the gastrointestinal tract (Outridge and Scheuhammer 1993). Because of its limited ability to permeate biological membranes, endocytosis by mucosal cells is the most likely means of entry of chromium(III) into the blood. In the blood, chromium(III) tends to bind to plasma proteins such as transferrin, with little penetration into erythrocytes. Transferrin-bound chromium(III) is rapidly cleared from the blood and appears mainly in the liver (reviewed by Cohen et al. 1993; Outridge and Scheuhammer 1993).

There is little evidence that biomagnification of chromium occurs in terrestrial food chains (Outridge and Scheuhammer 1993). Rather, chromium concentrations usually exhibit a pattern of "biominification" (reviewed by Outridge and Scheuhammer 1993).

Rats exposed to high concentrations of chromium oxide in their diets for more than 2 years showed no decreased body weight, food consumption, or life span, or histological abnormalities in major organs (Ivankovic and Presussmann 1975).

No adverse effects were observed in either mice or rats given 5 ppm chromium (III) as chromium acetate in drinking water throughout their life (Schroeder et al. 1964).

An LD_{50} of 60 mg for chromium (III) was established in rats injected intravenously (Mertz, 1975).

No information on the toxicological effects of chromium (III) on avian receptors was located.

D4-3-1.43 Chromium (VI) (CAS No. 7440-47-3)

As mentioned previously, chromium(VI) is generally more toxic than chromium(III). Although most chromium(VI) is reduced to chromium(III) in the acidic environment of the stomach (Donaldson and Barreras 1966), chromium(VI) compounds are absorbed significantly more efficiently from the gastrointestinal tract (2 to 10% of administered dose) than chromium(III) compounds (Outridge and Scheuhammer 1993). Once absorbed, chromium(VI) is quickly reduced to the trivalent form. The

damaging effects of chromium(VI) are due to its greater membrane permeability, which allows it to cross biological membranes and oxidize cellular components not normally accessible to chromium(III). As a result, the differences in systemic toxicity are primarily attributable to differential solubilities and absorption rates of the two valence states (Franchini and Mutti 1988).

Chromium(VI) compounds are absorbed significantly more efficiently from the gastrointestinal tract (2 to 10% of administered dose) than chromium(III) compounds. Once absorbed into the blood, chromium(VI) is rapidly taken up by erythrocytes via the general anion channel, and reduced to the trivalent from by various intracellular agents (e.g., glutathione, vitamins C and E, cytochrome P450, DT-diaphorase). Uptake and subsequent reaction appear to be similar in other cell types. Despite the rapidity of these uptake processes, chromium(VI)'s mobility and the limited supply of extracellular reductants causes it to be distributed more widely in the body than chromium(III). The intracellular reduction of chromium(VI) to chromium(III) generates unstable intermediate chromium(V) and chromium(IV) ions, active oxygen species (hydroxyl and superoxide radicals, singlet oxygen), and thiyl and organic radicals that are responsible for the cytotoxicity, mutagenicity, and carcinogenicity of the hexavalent form (reviewed by Manzo et al. 1992; Cohen et al. 1993; O'Flaherty 1993; Outridge and Scheuhammer 1993).

As noted above, chromium exhibits a pattern of biominification rather than biomagnification in ecological food webs. Because the speciation of chromium(VI) taken up by plants is poorly understood, it is assumed to be the primary form of exposure to herbivores. However, because chromium(VI) is immediately converted to chromium(III) in animal tissues, carnivorous receptors will be primarily exposed to the less toxic trivalent form.

Rosomer et al. (1961) (cited in 1986a) reported a subchronic NOAEL of 100 mg/kg in the diet for chickens.

Pregnant female mice receiving 250 mg/L potassium dichromate in drinking water throughout gestation showed no clinical signs of toxicity, but produced significantly fewer viable offspring (Trivedi et al. 1989). In the dog, 6 mg/L in drinking water (approximately 0.3 mg/kg-day) was a chronic NOAEL (Steven et al. 1976 [cited in Eisler 1986a]). A similar level was without observable effects in a study by Anwar et al. (1961).

The lethal single oral dose in young rats for chromium(VI) was established at 130 mg/kg (Samitz et al. 1962). Garner's Veterinary Toxicology (1967) has given the acute lethal dose for mature cattle at around 700 mg/kg, while 30 to 40 mg/kg of body weight produced poisoning in young calves. Inflamation and congestion of the stomach, ulceration of the rumen and abomasum, and high blood and liver chromium levels were characteristic findings.

D4-3-1.44 Chrysene (CAS No. 218-01-9)

Chrysene is absorbed by the oral route of exposure and may also occur following dermal exposure. No information was found in the available literature concerning effects of chrysene following oral exposure.

Intraperitoneal chrysene injections in male mice caused an increased incidence of liver tumors (Wislocki et al. 1986; Buening et al. 1979) and increased incidences of malignant lymphoma and lung tumors (Wislocki et al. 1986). In mouse skinpainting assays chrysene tested positive in both initiation and complete carcinogen studies (Wynder and Hoffman, 1959).

Male and female Swiss Webster BLU/Ha(ICR) mice received intraperitoneal injections of chrysene in DMSO (total dose = 320 ug/mouse) or DMSO alone on days 1, 8 and 15 after birth (Buening et al. 1979). Mice were killed at 38 to 42 weeks of age. The incidences of lung tumors in the treated group appeared to be elevated (5/24 (21%) and 1/11 (9%) in males and females, respectively), although not statistically significantly, when compared with the control groups (2/21 (10%) and 7/38 (18%) in males and females, respectively). The incidence of hepatic tumors in the treated males was statistically significantly greater (6/24, 25%) than in control males (0/21), whereas no hepatic tumors were found in the females. In a replication of this study, lung tumor incidence was not increased; however, the incidence of hepatic tumors in treated male mice was significantly elevated (6/27, 22%) over the incidence in the control group (0/52) (Chang et al. 1983). No liver tumors were reported in the females (IRIS 1994).

No information on the toxicological effects of chrysene on avian receptors was located.

D4-3-1.45 Cobalt (CAS No. 7440-48-4)

Cobalt is a dietary essential for ruminants and horses in which it is incorporated into vitamin B-12. Signs of cobalt deficiency in cattle and sheep are loss of appetite, body weight loss, emanciation, and anemia. Cobalt deficiency is more likely than cobalt toxicosis.

Environmental exposures to high levels of cobalt rarely occur. Characteristic signs of chronic toxicosis for most species are reduced feed intake and body weight, emanciation, anemia, hyperchromemia, debility, and increased liver cobalt (Turk and Kratzer, 1960).

A study by Brewer (1939) where cobalt was mixed with the food of dogs in amounts equivalent to 5, 10, 15, and 20 and 30 mgm at no time during the course of the four week study showed any toxic signs.

Adding cobalt in the form of cobalt chloride to the diet at levels up to 200 ppm did not result in toxicosis in pigs fed a diet adequate in iron (Huck and Clawson, 1976).

A study by Hill (1978) observed growth retardation and decreased resistance to infection in chicks fed cobalt in protein mixtures.

D4-3-1.46 Copper (CAS No. 7440-50-8)

Copper is widely distributed in nature and is an essential element for (1) the normal function of several critical enzymes and (2) the utilization of iron. Copper deficiency is, therefore, usually a greater health concern than copper excess. Copper absorption in the gastrointestinal tract is normally regulated by body stores. Absorbed copper is transported to the liver, where it may be incorporated into ceruloplasmin (a copper transport and donor molecule) and excreted into the plasma, stored as metallothionein or in lysosomes, or excreted via the bile (reviewed by Nederbragt et al. 1984).

Depressed food intake, body-weight gain, egg number and weight, and organ weights are associated with copper excess in poultry (Stevenson and Jackson 1981). The pair-feeding study was conducted to determine whether these effects were associated with direct toxicity or the accompanying marked reduction in food intake (Stevenson and Jackson 1981). Body weight, food intake, organ weights; egg production; egg weight; clinical chemistry parameters; and organ Cu, Fe, and Zn concentrations were monitored in laying hens fed varying concentrations of copper in their diet for 6

weeks (Stevenson and Jackson 1981). A NOAEL of 24 mg/kg/day was identified and used to develop TRVs for avian functional groups.

High doses of copper have caused liver and kidney damage as well as anemia in a number of species. It has been observed that the for stomach is also a target in rats and mice (Hebert et al. 1993). This well-designed subchronic feeding study examined histopathology, clinical pathology, reproductive toxicity, and tissue metal accumulation in males and females of both species.

An oral NOAEL was established in a chronic study of young calves (Cunningham 1946). The study confirms that young calves are susceptible to copper.

D4-3-1.47 Cyanide (CAS No. 57-12-5)

Many chemical forms of cyanide are present in the environment (e.g., metallocyanide, synthetic organocyanides, free CN). Only free cyanide is the primary toxic agent. Cyanides are readily absorbed through multiple pathways (inhalation, ingestion, dermal) and are rapidly distributed through the body. However, there are no reports of cyanide biomagnification or cycling in living systems. Cyanide is a potent and rapid asphyxiant. However, diagnosis of acute cyanide poisoning is often difficult because signs and symptoms are nonspecific and toxicity is influenced by a variety of factors. Deficiencies of vitamin B12, iodine, and sulfur amino acids can modify its biocidal properties.

In a 2-year dietary study (Hartung, 1982), rats (10/sex/group) were administered food fumigated with HCN. The average daily concentrations were 73 and 183 mg CN/kg diet. From the data reported on food consumption and body weight, daily estimated doses were 4.3 mg and 10.8 mg CN/kg bw. The average food CN concentrations were estimated based on the authors' data for concentration at the beginning and end of each food preparation period and by assuming a first-order rate of loss for the intervening period. There were no treatment related effects on growth rate, no gross signs of toxicity, and no histopathologic lesions.

A rat chronic oral toxicity study (Howard and Hanzal, 1955; EPA, 1984) indicated a NOAEL of 20.4 mg/kg-day. This 2-year study showed no signs of cyanide toxicity.

D4-3-1.48 Diethyl phthalate (CAS No. 84-66-2)

Groups of CD rats (15/sex) were fed diets containing 0, 0.2, 1.0, or 5.0% DEP for 16 weeks. The authors estimated the mean intakes to be 0, 150, 770, and 3160 mg/kg/day for the males and 0, 150, 750, and 3710 mg/kg/day for the females. Additional groups of five rats/sex were fed similar diets for 2 or 6 weeks. Hematological examinations (red blood cell count, hematocrit, hemoglobin) were performed on animals fed diets for 2, 6, and 16 weeks. Differential white blood cell counts were also conducted on 0 and 5% dose groups at 16 weeks. Food and water intake and body weight were measured for all groups weekly. Urinalyses were conducted during weeks 2, 6, and 15 on 5 to 15 rats/sex/dose group. After 16 weeks of treatment, autopsy, hematologic and histologic examinations were conducted on all animals. No changes in behavior or other clinical signs of toxicity were observed. The authors reported significantly less weight gain throughout the duration of the experiment in both sexes given 5% DEP (15 to 25% decrease) and in females (5 to 8% decrease) fed 1% DEP. Mean food consumption of the previous groups was also decreased (by 11 to 23%) relative to controls. No significant dose- or time-related trends in urinalysis or hematology results were found. Absolute weights of brain, heart, spleen, and kidneys were decreased in both sexes fed 5% DEP. Relative weights of the brain, liver, kidneys, stomach, small

intestines, and full caecum were significantly greater in both sexes after 16 weeks at the 5% dietary level when compared with controls.

In another experiment, groups of six rats/sex were pair-fed diets containing either 0 or 5% DEP for 16 weeks. Body weights were measured weekly. The authors reported that rats fed 5% DEP consumed more food and gained less weight than controls. The differences in food consumption (1 to 5%) were not statistically significant, and mean weight differences were 7 to 10%, which the authors reported as statistically significant.

A 2-year feeding study using rats was conducted by Food Research Laboratories, Inc. in 1955. Albino weanling rats (strain not specified) (15/sex) were fed 0, 0.5, 2.5, and 5.0% diethyl phthalate in the diet. Animals were maintained for a 2-year period during which two males and two females/group were examined at 12-week intervals for the following: red and white blood cell counts, differential white count, hemoglobin, blood sugar and nitrogen, and urinalysis. Growth of animals in the 5% treatment group was retarded throughout the study, with no depression of food intake. There was a significant decrease in efficiency of food utilization in this group compared with controls. There were no other treatment-related effects either on the parameters listed above or on gross organ appearance or histopathology (IRIS 1994).

Data regarding developmental and reproductive effects is extremely limited. Singh et al. (1972) observed skeletal malformations in Sprague-Dawley rats after i.p. administration (0.506, 1.012, and 1.686 mL/kg) on days 5, 10, and 15 of gestation. In addition, fetuses were significantly smaller than untreated controls. Exposure to DEP does not appear to affect the reproductive performance of mice after oral administration of 0.25, 1.25, and 2.5% DEP for 18 weeks (NTP, 1984). Second-generation breeding pairs exposed to 2.5% DEP exhibited increased right epididymis and prostate weights in males and decreased pituitary weight in females (NTP, 1984).

D4-3-1.49 Di-n-butyl phthalate (CAS No. 84-74-2)

No data on the carcinogenicity of di-n-butyl phthalate are available. Di-n-butyl phthalate exhibits low acute toxicity in laboratory animals. Rats injected with di-n-butyl phthalate exhibited decreased birth weight, increased embryo mortality, and teratogenic effects (skeletal abnormalities) (Singh et al. 1972). Oral administration of 2000 mg/kg-day for nine days caused sever seminiferous tubular atrophy in rats and guinea pigs (Federal Register, 1983).

Smith (1953) fed male rats in groups of 10 per dose 0, 0.01%, 0.05%, 0.25%, and 1.25% di-n-butyl phthalate for one year. A dose of 125 mg/kg-day was derived from a figure depicting daily intake in Smith (1953). More than one-half of the rats in the highest treatment group died during the first week of exposure. The remaining animals survived with no apparent ill effects. A NOAEL of 125 mg/kg-day was established.

Di-n-butyl phthalate has been demonstrated to be toxic to fetuses in a number of animal studies. Administration of di-n-butyl phthalate during pregnancy at doses of 2,100 mg/kg/day resulted in reduced fetal weight and a decreased number of viable litters in mice (Hardin et al. 1987).

High doses of di-n-butyl phthalate appear to have an adverse effect on reproduction in female animals. Pregnant rats or mice fed 7,500 to 10,000 ppm of di-n-butyl phthalate in the diet during gestation experienced marked decreases in the number of pups that were born alive (Killinger et al. 1988).

D4-3-1.50 Di-n-octylphthalate (CAS No. 117-84-0)

There is limited toxicity information on this compound. This compound is reported to be mildly toxic by ingestion, and is a severe skin and eye irritant under acute exposure.

No information on the toxicological effects of di-n-octylphthalate on avian receptors was located. No other information for the toxicity of di-n-octylphthalate was found.

D4-3-1.51 Di-2-ethylhexyl-phthalate (DEHP) (CAS No. 117-81-7)

The following numbers of guinea pigs were fed diets containing DEHP for a period of 1 year: 24 males and 23 females consumed feed containing 0.13% DEHP, 23 males and 23 females consumed feed containing 0.04% DEHP, and 24 males and 22 females were fed the control diet. These dietary levels corresponded to 64 or 19 mg/kg bw/day based on measured food consumption. No treatment-related effects were observed on mortality, body weight, kidney weight, or gross pathology and histopathology of kidney, liver, lung, spleen, or testes. Statistically significant increases in relative liver weights were observed in both groups of treated females (64 and 19 mg/kg bw/day).

Groups of 32 male and 32 female Sherman rats were maintained for 2 years on diets containing either 0.04, 0.13 or 0.4% DEHP (equivalent to 20, 60, and about 195 mg/kg bw/day based on measured food consumption). An F1 group of 80 animals was fed the 0.04% diet for 1 year. Mortality in the F1 treated and control groups was high, 46.2 and 42.7%, respectively, survived to 1 year. There was, however, no effect of treatment on either parental or F1 group mortality, life expectancy, hematology, or histopathology of organs. Both parental and F1 rats receiving the 0.4% DEHP diet were retarded in growth and had increased kidney and liver weights.

In an NTP (1982) study, 50 male and 50 female Fisher 344 rats per group were fed diets containing 0, 6,000 or 12,000 ppm DEHP for 103 weeks. Similarly, groups of 50 male and 50 female B6C3F1 mice were given 0, 3,000 or 6,000 ppm DEHP in the diet for 103 weeks. Animals were killed and examined histologically when morbund or after 105 weeks. No clinical signs of toxicity were observed in either rats or mice. A statistically significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma were observed in female rats and both sexes of mice.

It appears that guinea pigs offer the more sensitive animal model for DEHP toxicity. A LOAEL in this species is determined to be 19 mg/kg/day (IRIS 1994).

No information on the toxicological effects of DEHP on avian receptors was located.

D4-3-1.52 Ethyl Alcohol (Ethanol) (CAS No. 64-17-5)

The effects of ethanol in animals is well studied. The critical effect with exposure to ethanol is related to the heart. One study of the effects from ingestion of ethanol by rats over 6-, 12-, and 18-months indicates the densities of cells on the left ventricle of the heart are less than the controls when fed 9 g/kg-d (Cadete-Leite et al. 1989). This effect is not reversible. No significant difference in body weight was observed. Young rats exposed to 6.6 g/kg of ethanol each day between postnatal days 4 and 10 and the number of Purkinjie cells in the granular layer of the heart were significantly reduced (Pierce, et al. 1989).

Mankes et al. (1982) observed that at 0.4 mL/kg-d through gestation had no effect on most reproductive parameters and the incidence of malformed fetuses was significantly increased at this dose level. This dose level was therefore considered to be a LOAEL.

Dogs fed a diet mixed with 3 g/kg ethanol for 3 months and 9 months had similar heart problems (Horton, 1989). These problems include altered cardiocirculatory function with increased coronary blood flow, myocardial oxygen delivery, extraction, and consumption and an increased total myocardial tissue calcium content. Pregnant dogs fed 500 mL/d in their diets for 20 weeks from the first day of pregnancy induced slight but discernible changes in the central nervous system of the pups including neuronal damage within the frontal and hippocampal cortex and Purkinje cell layer and decreased level of cholesterol esters as compared to controls (Marciniak et al. 1974).

Acute exposure to ethanol in monkeys (2.5 and 4.1 g/kg once weekly from 40 days gestation to term) causes neurologic and developmental anomalies although less severe at the lower dose (Shepard 1986).

Inhalation of ethanol by guinea pigs at 6,400 ppm did not cause any signs of intoxication (Verschueren 1983).

D4-3-1.53 Ethylbenzene (CAS No. 100-41-4)

No data on the carcinogenicity of ethylbenzene was located. Rats and rabbits exposed to 1,000 ppm PCE as a vapor seven hours a day for three weeks during pregnancy did not exhibit maternal or fetal toxicity. An Oral LD_{50} value for rats of 3,500 mg/kg has been reported. Chronic inhalation of 400 to 2,200 ppm PCE for six months produced no adverse effects in guinea pigs, monkeys, and rabbits (Wolf et al. 1956). Liver and kidney effects were observed in rats given 408 and 680 mg/kg-day ethylbenzene in olive oil for six months (Wolf et al. 1956).

Ten adult albino rats were given doses of ethylbenzene in olive oil for 182 days. Treatment levels were 0, 13.6, 136, 408, and 680 mg/kg-day. Criteria for judging toxic effects in rats were growth, mortality, appearance, behavior, hematologic changes, terminal concentration of urea nitrogen in the blood, and bone marrow counts. A LOAEL of 408 mg/kg-day is associated with histopathologic changes in the liver and kidney (Wolf et al. 1956).

No information on the toxicological effects of ethylbenzene on avian receptors was located.

D4-3-1.54 Fluoranthene (CAS No. 206-44-0)

Male and female CD-1 mice (20/sex/group) were gavaged for 13 weeks with 0, 125, 250, or 500 mg/kg/day fluoranthene. A fifth group of mice (30/sex) was established in the study for baseline blood evaluations. Body weight, food consumption, and hematological and serum parameter values were recorded at regular intervals during the experiment. At the end of 13 weeks, the animals were sacrificed and autopsied, which included organ weight measurement and histological evaluation. All treated mice exhibited nephropathy, increased salivation, and increased liver enzyme levels in a dose-dependent manner. However, these effects were either not significant, not dose-related, or not considered adverse at 125 mg/kg/day. Mice exposed to 500 mg/kg/day had increased food consumption and increased body weight. Mice exposed to 250 and 500 mg/kg/day had statistically increased SGPT values and increased absolute and relative liver weights. Compound-related microscopic liver lesions (indicated by pigmentation) were observed in 65 and 87.5% of the mid- and high-dose mice, respectively. Based on

increased SGPT levels, kidney and liver pathology, and clinical and hematological changes, the LOAEL is considered to be 250 mg/kg/day, and the NOAEL is 125 mg/kg/day (IRIS 1994).

Fluoranthene has been tested as a complete carcinogen in mouse skin-painting assays at doses ranging approximately from 1.5 mg/mouse/week for 52 weeks to 100 mg/mouse/week for 82 weeks; the results of these studies have been consistently non-positive (Suntzeff et al. 1957; Wynder and Hoffmann, 1959; Hoffmann et al. 1972; Horton and Christian, 1974).

D4-3-1.55 Fluorene (CAS No. 86-73-7)

Adult male and female mice (25 each sex/treatment group) were given oral (corn oil gavage) doses of fluorene for 13 weeks. Treatment levels were 0, 125, 250, and 500 mg/kg-day. Toxicological evaluations included body weight changes, food consumption changes, mortality, clinical pathology, organ weights, and histopathological evaluations of target organs. A significant decrease in red blood cell count, packed cell volume, hemoglobin concentration, and total serum bilirubin levels was in females from 250 mg/kg-day treatment group (EPA, 1989). A LOAEL of 250 mg/kg-day was established.

Morris et al. (1960) fed female buffalo rats a diet containing 0.05% fluorene in 3% corn oil for approximately 18 months or in propylene glycol for about 6 months (approximately 11 mg/kg/day). Various tumors occurred in controls and exposed animals at approximately the same incidences, ranging from 6 to 34%.

D4-3-1.56 Fluoride (CAS No. 16984-48-8)

Inorganic fluorides are generally highly irritating and toxic. Acute effects resulting from exposure to fluorine compounds are due to HF. Chronic fluorine poisoning, or fluorosis, occurs among numbers of cryolite, and consists of a sclerosis of the bones caused by a fixation of the calcium by the fluorine. There may also be some calcification of the ligaments. The teeth are mottled, and there is osteosclerosis and osteomalacia. Large doses can cause very severe nausea, vomiting, diarrhea, abdominal burning and cramp-like pains. Fluoride is not taken up by the thyroid and does not interfere with iodine uptake. It can cause or aggravate attacks of asthma and severe bone changes, making normal movements painful. Some signs of pulmonary fibrosis have been noted (Sax and Lewis 1987).

The reproductive effects of fluoride administered orally in the diet of minks was studied (Aulerich et al. 1987). Five dose levels were administered. Fluoride up to 229 ppm had no adverse effects on reproduction. Survivorship of kits in the 385 ppm group was significantly reduced. These doses were considered to be NOAELs and LOAELs, respectively. Because the study considered exposure over 382 days including critical life stages (reproduction), these doses were considered to be chronic. A NOAEL of 31.37 mg/kg/d was established.

The effects of fluoride administered to the screech owl orally in the diet for a period of 5 to 6 months were studied (Pattee et al. 1988). The fertility and hatching success were significantly reduced by 9U232 ppm F the diet, 56.5 ppm F in the diet had no adverse effect. Because the study considered exposure during reproduction, these doses were considered to be chronic. A NOAEL of 7.8 mg/kg/d was established and used to develop a TRV (IRIS 1994).

D4-3-1.57 Formaldehyde (CAS No. 50-00-0)

Formaldehyde has been identified as a carcinogen in humans (Blair et al. 1986) and there is sufficient data in mice and rats that formaldehyde also causes cancer in these animals from inhalation exposures (Kerns et al. 1983). Squamous cell carcinomas of the nasal cavity were observed in mice at doses as low as 5.6 ppm.

Formaldehyde administered in drinking water to rats for two years (Til et al. 1989) indicates a decrease in both food and water intake, decreases in heart and liver weights in males, and increases in brain and kidney weight for females. Three dose groups were used in the study and they are 1.2, 15, and 82 mg/kg-d for males and 1.8, 21, and 109 mg/kg-d for females. At the higher dose levels thickening of the forestomach and glandular stomach were observed with ulceration of the stomach.

The LD_{50} for formaldehyde was shown by Skog (1950) to be 300 mg/kg in mice, and 420 mg/kg in rats following sc injections of 150 to 460 mg/kg in 72 mice and 300 to 640 mg/kg in 64 rats.

Reproductive effects have been studied in Beagle dogs (Hurni and Ohder 1973) at a dose of 9 mg/kg-d with no effects observed and in mice (Marks et al. 1980) at 148 mg/kg-d with no effects observed (IRIS 1994).

D4-3-1.58 Hydrazine (CAS No. 302-01-2)

Toxic effects such as respiratory tract irritation, hemolytic anemia, kidney and liver damage, central nervous system damage, and tumorigenic effects have been observed in experimental animals.

Rothberg and Cope measured the acute toxicity of hydrazine by the intravenous route. Rabbits were injected and iv and observed for 24 hours. The LD_{50} by the iv route was 26 mg/kg.

A study of the effect of hydrazine on pregnant rats was described by Lee and Aleyassine in 1970. Wistar rats received hydrazine at 8 mg/kg/day for 10 days. No offspring survived the first 24 hours after birth. The offspring were pale and edematous, with occasional petechial hemorrhages.

Biancifiori (1970) conducted a multiple-dose study in which hydrazine sulfate was administered by gavage to groups of 24 to 30 8-week-old CBA/Cb/Se mice of each sex at doses of 0.0, 0.14, 0.28, 0.56, or 1.13 mg/day, 6 days/week for 25 weeks. Animals were observed throughout their lifetimes. Liver carcinomas were induced in a dose-related manner in both sexes and lung metastases were observed in some of the mice treated with 1.13 mg/kg/day. Pulmonary tumors were reportedly present in many of the treated mice, but incidences were not reported because the purpose of the study was to describe hepatic tumors.

Many other water gavage studies of hydrazine sulfate in mice have resulted in increased incidence of lung adenomas/carcinomas. Strains tested included BALB/c (Biancifiori and Ribacchi, 1962), CBA/Cb/Se (Biancifiori et al. 1964; Severi and Biancifiori, 1968), BALB/c x DBA/2 (Kelly et al. 1969) and Swiss (Roe et al. 1967). Hepatomas and hepatocarcinomas were also observed in some strains as a consequence of treatment. Cb/Se rats gavaged with 18 (males) or 12 (females) mg hydrazine sulfate/day showed an increased incidence of lung tumors in both sexes and hepatomas in males only (Severi and Biancifiori, 1968).

Toth (1969) administered 0.012% hydrazine sulfate in the drinking water to groups of 6-week old Swiss, C3H, and AKR mice (40 to 50/sex) for their lifetimes. Groups of 110 Swiss mice and 30 C3H and AKR mice of each sex served as untreated controls. Lung adenomas and adenocarcinomas were reported in 46-50% of the treated Swiss mice (25/50 males and 24/50 females), compared with 9 to 11% in the controls (11/110 males and 14/110 females). Hydrazine sulfate did not induce significantly increased incidence of tumors at other sites in the Swiss mice, or at any site in the C3H or AKR mice. In a later study, Toth (1972) administered 0.001% hydrazine continuously in the drinking water to 50 Swiss mice/sex for their lifetimes. Lung adenomas and adenocarcinomas were induced in 24/50 of the males and 27/50 of the females (48 to 54%). Yamamoto and Weisburger (1970) reported a 100% induction of lung adenomas and adenocarcinomas (38/38 by comparison with 12/20 in the controls) in A/J male mice given 325 mg/L hydrazine sulfate in the drinking water for 48 weeks.

MacEwen et al. (1981) reported on the carcinogenic effect of inhaled hydrazine in C57BL/6 mice, F344 rats, Syrian golden hamsters and beagle dogs. Hydrazine vapor (97% pure) was administered to 400 female mice at 0.05, 0.25 or 1.0 ppm; to 100 rats of each sex at 0.05, 0.25, 1.0, or 5.0 ppm; to 160 male hamsters at 0.25, 1.0 or 5.0 ppm; and to 4 dogs of each sex at 0.25 or 1.0 ppm. Exposure was 6 hours/day, 5 days/week for 1 year, followed by a variable observation period (12 to 38 months). Appropriate controls were maintained for each species. Significantly increased incidences of tumors were reported at the highest exposures administered in mice (lung adenoma), male and female rats (nasal cavity adenoma and adenocarcinoma), and hamsters (nasal cavity polyp) as well as in male and female rats treated with 1.0 ppm hydrazine (nasal cavity adenoma and adenocarcinoma). No significant increase in tumor induction was observed at the lower doses nor were treatment-related neoplasms reported in the dogs. The observation period is considered to be insufficient for dogs.

Juhasz et al. (1966) injected white mice of both genders with hydrazine (0.5 mg x 16 injections) over a period of 46 days, then observed the animals for 1 year. Mediastinum reticulum-cell sarcomas were observed in 4/34 mice, and 9/34 had myeloid leukemias. A single thymic leukemia was reported out of 60 control animals. Kelly et al. (1969) injected (BALB/c\x DBA/2)F1 male mice i.p. with a total dose of 20.8 mg hydrazine sulfate/animal (given in 8 weekly injections). Lung tumors were reported in 6/30 of the treated animals and 1/9 of the control animals (IRIS 1994).

D4-3-1.59 Hydrofluoric acid (HF) (CAS No. 7664-39-3)

Potential dangers associated with exposure to HF solutions depends upon the concentration. A 20% solution caused immediate damage to experimental rabbits. Exposure to HF via inhalation can also be toxic, causing eye and respiratory tract irritation and damage.

A LC_{50} of 342 ppm was reported for mice exposed to HF via inhalation for one hour. Toxic effects included corneal damage and other eye effects (J. Combust. Toxicol., 3:61, 1976; RTECHS). A TCL_O of 470 ug/m3 was reported for rats exposed to HF via inhalation four hours a day days one through 22 of pregnancy. Adverse effects on fertility were observed, including pre- and post-implantation mortality (Gig. Tr. Prof. Zarbol. 19(3):57, 1975; RTECHS). No other information for the toxicity of hydrofluoric acid was found.

D4-3-1.60 Isophorone (CAS No. 78-59-1)

Beagle dogs (4/sex/dose) were administered gelatin capsules containing 0, 35, 75, or 150 mg isophorone/kg/day 7 days/week for 90 days (Nor-Am Agricultural Products, 1972). All dogs survived the study in good condition. Food consumption was within normal limits and body weight was not affected by treatment. All organs appeared normal at gross examination and no significant changes in organ

weight were produced with the ingestion of isophorone. There were no definitive signs of cellular change in any of the tissues examined. A NOEL for systemic toxicity was established at 150 mg/kg/day (HDT) due to the lack of effects produced at any dose tested.

NTP (1984) administered 0, 250, or 500 mg isophorone/kg/day, 5 days/week for 103 weeks by corn oil gavage to groups of 50 F344/N rats/sex and 50 B63F1 mice/sex (averaged daily doses: 0, 179, and 357 mg/kg/day). Doses selected for the 2-year studies were based on 16-day repeated-administration studies in which rats and mice of each sex received 0 to 2,000 mg/kg/day and on 13-week studies in which rats and mice of each sex received doses ranging from 0 to 1,000 mg/kg/day by corn oil gavage.

Dosed male rats showed a variety of proliferative lesions of the kidney (tubular cell hyperplasia, 0/50, 1/50, 4/50; tubular cell carcinoma, 0/50, 0/50, 2/50; tubular cell adenocarcinoma, 0/50, 3/50, 0/50; and epithelial hyperplasia of the renal pelvis, 0/50, 5/50, 5/50 in the 0, 250, and 1,000 mg/kg/day, respectively). Dosed male rats also exhibited increased mineralization of the medullary collecting ducts (1/50, 31/50, 20/50 in the 0, 250, and 1,000 mg/kg/day, respectively), and male rats receiving 250 mg/kg/day showed a more severe nephropathy than is commonly seen in aging F344/N rats. With the exception of a moderate increase in nephropathy, female rats did not show chemically related increased incidences of neoplastic or nonneoplastic lesions (IRIS 1994).

No reliable studies were located regarding the systemic effects in animals following acute oral exposure to isophorone. No studies were locted regarding developmental effects or reproductive effects following acute oral exposure to isophorone. The study by Nor-Am Agricultural Products (1972) appears to be the best available study of the affects of isophorone on animals. A NOAEL of 150 mg/kg-day was established from this study.

D4-3-1.61 Lead (CAS No. 7439-92-1)

Lead is a ubiquitous trace constituent in rocks, soils, plants, water, and air, with an average concentration of 16 mg/kg in the earth's crust (Eisler 1988b). Lead has four stable isotopes: Pb-204 (1.5%), Pb-206 (23.6%), Pb-207 (22.6%), and Pb-208 (52.3%). Lead occurs in four valence states: elemental (Pb°), monovalent (Pb^{+}), divalent (Pb^{+2}), and tetravalent (Pb^{+4}). In nature, lead occurs mainly as Pb^{+2} and is oxidized to Pb^{+4}. Metallic lead is relatively insoluble in hard waters. Some lead salts are somewhat soluble in water. Of the organoleads, tetraethyllead and tetramethyllead are the most stable and are highly soluble in many organic solvents but are fairly insoluble in water. Both undergo photochemical degradation in the atmosphere to elemental lead and free organic radicals. Organolead compounds are primarily anthropogenically-produced (Eisler 1988b).

Lead is neither essential nor beneficial to living organisms. Lead affects the kidney, blood, bone, and central nervous system. Effects of lead on the nervous system is both functional and structural. Lead toxicity varies widely with the form and dose of administered lead. In general, organolead compounds are more toxic than inorganic lead. In nature, lead occurs mainly as divalent, Pb^{2+}. Ingestion of lead shot by regulatory waterfowl is a significant cause of mortality in these species.

Chicken, quail, and pheasant hatchlings are relatively tolerant to moderate lead exposure (Eisler 1988b). There was no effect on hatchling growth of these species at dietary levels of 500 mg/kg or on survival to 2,000 mg/kg lead (Hoffman et al. 1985 as cited in Eisler 1988b). Altricial species are generally more sensitive to lead than precocial species (Eisler 1988b) of avian insectivores. American Kestrels (Falco sparverius) exposed to 50 mg/kg/day metallic lead in diets did not exhibit effects on survival or reproductive success (Colle et al. 1980).

D4-3-1.62 Magnesium (CAS No. 7439-95-4)

Magnesium is an essential nutrient for living organisms, playing a critical role in cell metabolism and is essential for neuromuscular contraction. The gastrointestinal absorption of magnesium depends upon the species, dose, and solubility of the magnesium compound involved. The absorption of dietary magnesium is about 15% in dogs and rabbits, 40% in cows, and 60% in rats. Excretion of magnesium is both fecal and urinary. Magnesium does not exist in a pure state in nature but exists mainly as magnesite, carnallite, and dolomite. Only with impaired kidney function could large doses of oral doses of magnesium cause toxicity. Magnesium toxicity is usually acute. Symptoms include nausea, muscular weakness, general paralysis, and paralysis of respiratory, cardiovascular, and central nervous systems. Magnesium salts are generally non-toxic when administered orally.

Ingestion of excess levels of magnesium has generally resulted in decreased growth rates in chicks (Nugara and Edwards, 1963), guinea pigs, and sheep. Generally, the high intake of magnesium increases blood serum magnesium.

Intravenous infusion of high levels of magnesium ions resulted in disruption of motor function in horses, cattle, and dogs (Bowen et al. 1970). The levels required to produce the effect were 0.13 to 0.14 g magnesium sulfate per kilogram of body weight.

A MLD was reported for dogs exposed orally to 230 mg/kg magnesium. Fifty percent of rats given 715 mg (as magnesium chloride) died (LD_{50} = 715 mg/kg). A MLD dose of 390 mg/kg was reported for guinea pigs given magnesium fluoride orally (Venugopal and Luckey, 1978).

Twelve 3- to 6-year old sheep were exposed to 4 mg/kg MgCl in drinking water for 16 months. A lower feed intake and diarrhea resulted (Pierce, 1959; EJM).

D4-3-1.63 Manganese (CAS No. 7439-96-5)

The bioavailability of different forms of manganese varies considerably depending on different exposure conditions. There is potentially higher bioavailability of manganese from drinking water than food. It is also important to recognize that various dietary factors as well as the form of manganese can have a significant bearing on the dose absorbed from the gastrointestinal tract. For instance, many constituents of a vegetarian diet (e.g., tannins, oxalates, phytates, fiber, calcium, and phosphorus) have been found to inhibit manganese absorption presumably by forming insoluble complexes in the gut. Thus, herbivores are more likely to be resistant to manganese toxicity. Also, the form of manganese can significantly influence toxicity. For example, mice receiving the two soluble forms of manganese (chloride and acetate salts) were found to gain significantly less weight than controls, while mice consuming the insoluble forms of manganese (carbonate and dioxide salts) appeared to actually gain slightly more weight than controls.

DiPaolo (1964) subcutaneously or intraperitoneally injected DBA/1 mice with 0.1 mL of an aqueous of solution 1% manganese chloride twice weekly for 6 months. A larger percentage of the mice exposed subcutaneously (24/36; 67%) and intraperitoneally (16/39; 41%) to manganese developed lymphosarcomas compared with controls injected with water (16/66; 24%). In addition, tumors appeared earlier in the exposed groups than in the control groups. The incidence of tumors other than lymphosarcomas (i.e., mammary adenocarcinomas, leukemias, injection site tumors) did not differ significantly between the exposed groups and controls.

A study reporting the minimum manganese requirements in chickens was used to derive a TRV of 2.9 mg/kg/day. Guinea fowl were found to have reduced hatchability and increased deformed embryos when fed diets deficient in manganese (Offiong and Abed 1980).

For rats, the estimated requirement is 50 mg Mn/kg diet (Rogers 1979). A dietary reproduction study in rats exposed to 250 ppm manganese (13 mg/kg/day) was used to develop a TRV of 1.1 mg/kg/day (Laskey et al. 1982).

D4-3-1.64 Mercury (CAS No. 7439-96-5)

Mercury exists in the environment in three oxidation states: the element itself, + 1 (mercurous) state, and + 2 (mercuric) state. Although the generally more toxic organic forms of mercury are unlikely to persist in the environment, they (in particular, methylmercury) may be formed in biotic tissues and are known to biomagnify through ecosystems, particularly aquatic systems (reviewed by Wren 1986; Scheuhammer 1987).

Because of its chemical stability and lipophilicity, methylmercury readily penetrates the blood-brain barrier. The central nervous system is thus a major target organ in both mammals and birds. However, reproductive effects have been reported at even lower doses. Methylmercury can be converted to inorganic mercury both in tissues and by microflora in the gut. The homolytic cleavage of the mercury-carbon bond leads to generation of reactive intermediates, e.g., methyl and metal radicals, which cause cellular damage (reviewed by Wren 1986; Scheuhammer 1987; Manzo et al. 1992).

The effects of mercury on avian herbivores, insectivores, and carnivores were evaluated as follows. For herbivores, the effects of organic mercury compounds on galliformes (domestic chickens, quail, pheasants) have been investigated by several groups. However, no study was reviewed that identified an NOAEL. The lowest LOAEL for relevant endpoints (reproductive success) of several similar studies was found in a study of the effects of mercury to birds (Fimreite 1979). Reduced egg production, shell thickness, and hatchability in pheasants fed seed treated with organomercurial fungicide were observed.

Three goshawks were fed a diet of chickens that had eaten wheat dressed with an organomercurial fungicide (Borg et al. 1970). Their tissues contained 10 to 40 ppm of mercury, mostly as methylmercury. The hawks died after 30 to 47 days; their total mercury intake was about 20 mg/bird.

Two studies examined the effects of subchronic methylmercury exposure on the reproductive competence of male and female rats (Khera and Tabacova 1973; and Khera 1973). The NOAEL identified for both sexes was 0.25 mg/kg/day. Much less information is available regarding methylmercury toxicity to herbivores.

In a study of acute methylmercury toxicity in mule deer (Odocoileus hemionus hemionus) 17.88 mg/kg was said to be the LD_{50} (Eisler 1987a). A number of studies have examined the effects of chronic methylmercury ingestion on carnivorous mammals, particularly cats (e.g., Albanus et al. 1972; Charbonneau et al. 1976; Eaton et al. 1980) and mink (e.g., Aulerich et al. 1974; Wobeser et al. 1976; Wren et al. 1987). The chronic toxicity of cats study by was considered superior to other available studies because of its long duration (2 years), use of relatively large group sizes, detailed examination of endpoints, identification of both no-effect and effect levels, and administration of mercury via both contaminated fish and addition to diet (Charbonneau et al. 1976).

D4-3-1.65 Methyl Alcohol (Methanol) (CAS No. 67-56-1)

Methanol is a poison by ingestion, skin contact, intravenous, and intraperitoneal routes. It is moderately toxic by inhalation and other routes and is an eye and skin irritant, a systemic irritant by inhalation, and a narcotic. Once absorbed, methanol is only very slowly eliminated and, in the body, the products formed by its oxidation are formaldehyde and formic acid, both of which are toxic. It is regarded as a cumulative poison (Sax and Lewis, 1987).

Sprague-Dawley rats (30/sex-dose) were gavaged daily with 0, 100, 500, or 2500 mg/kg-day of methanol (US EPA, 1986). This 90-day, subchronic test suggests possible treatment-related effects in rats dosed with 2500 mg methanol/kg-day by elevated levels of SGPT, SAP, and increased, but not statistically significant, liver weights in both male and female rats, despite the absence of supportive histopathologic lesions in the liver. Brain weights of both high-dose group males and females were significantly less than those of the control group. Based on these findings, 500 mg/kg-day of methanol is considered a NOAEL in rats (IRIS 1994).

No information on the toxicological effects of methanol on avian receptors was located.

D4-3-1.66 Methyl isobutyl ketone (CAS No. 108-10-1)

The majority of the available literature for methyl isobutyl ketone is on the inhalation exposure pathway. One study that is available on the ingestion exposure pathway is a 13-week study on rats given a dose of 250 mg/kg-d (Microbiological Associates, 1986). No adverse effects were observed so this value is a NOAEL. However, increased relative and absolute weights of the liver and kidney were observed in females, and increased lethargy and urinary protein levels were also observed in females. Another oral study on mice and rats (Batyrova 1973) indicates that the lethal doses of methyl isobutyl ketone are 2.85 and 4.6 g/kg, respectively.

Several studies have been conducted that address the effects of inhalation of methyl isobutyl ketone on animals. These studies indicate that approximately 25 ppm for 24-hr for seven days causes hand to eye coordination problems in juvenile baboons (Geller et al. 1979), whereas 100 ppm in a continuous exposure for 90-days to monkeys, rats and dogs produced no significant changes in clinical chemicals or hematology (ACGIH 1986). Another study on exposure to rats (Patty 1981-1982) showed a minimal statistical increase in pressor lever response. For baboons, 20—40 ppm caused no problems in behavior response time. Higher concentrations exposures for rats (i.e., 100-200 ppm) causes increases in kidney and liver weights with reversible kidney damage after exposure for 2 weeks to 100 ppm.

D4-3-1.67 Methylene chloride (dichloromethane) (CAS No. 75-09-2)

Inhaled methylene chloride is metabolized to carbon dioxide in animals leading to the formation of carboxyhemoglobin and subsequent oxygen deprivation.

Studies in animals confirm that methylene chloride may be lethal after inhalation exposure at high concentrations. Acute exposure to 16,000-19,000 ppm methylene chloride for 4 to 8 hours caused death in rats and mice (NTP 1986).

Four groups of 85 each male and female rats were fed 5, 50, 125, and 250 mg/kg-day for 2 years (National Cancer Association, 1982). Many effects were evaluated. Treatment-related histological alternations of the liver were evident at the low dose of 50 mg/kg-day. No adverse effects were observed

at the 5 mg/kg-day level. An LD_{50} value of 2,100 mg/kg was reported in rats by ingestion (Kimura et al. 1971).

Studies in animals provide suggestive evidence that ingestion of methylene chloride may increase the risk incidence of liver cancer. Liver tumors were observed in female but not in male rats that ingested methylene chloride (up to 250 mg/kg/day) for 2 years (Serota et al. 1986).

No information on the toxicological effects of methylene chloride avian receptors was located.

D4-3-1.68 Molybdenum (CAS No. 7439-98-7)

Molybdenum exposure can occur through inhalation of dust or fumes, ingestion, or eye and skin contact for soluble compounds. It is considered to be an essential trace element in many species, including man. Animal studies indicate that insoluble molybdenum compounds are of a low order of toxicity. Freshly generated molybdenum fumes, however, are considerably more toxic. Inhalation of high concentrations of molybdenum trioxide dust is very irritating to animals and has caused weight loss, diarrhea, loss of muscular coordination, and a high morality rate. Molybdenum trioxide dust is more toxic than fumes. Large oral doses of ammonium molybdate in rabbits caused some fetal deformities. Excessive intake of molybdenum may produce signs of a copper deficiency (Sittig 1981).

The results of three studies (TOXNET 1994) on the toxicological effects of molybdenum on rats and guinea pigs described LOAELs of 2, 7.5, and 40 mg/kg-day. The most conservative of these was used to develop a TRV of mammalian receptors.

Information on the toxicological effects of molybdenum on avian function groups was not located.

D4-3-1.69 Naphthalene (CAS No. 91-20-3)

Oral and subcutaneous administration of naphthalene did not cause cancer in rats. Intraperitoneal injection of 395 mg/kg-day naphthalene in rats did not cause adverse fetal or maternal effects (Hardin et al. 1981). Ocular toxicity is the most common effects associated with short-term, high-level exposures in animals. Rabbits fed 1000 mg/kg-day for 46 days experienced cataracts and retinopathy (Ghetti and Mariana, 1956). Bronchial necrosis was observed in mice following a single intraperitoneal injection of 128 mg/kg (Mahavi et al. 1977). Dogs experienced a 98% decrease in hemoglobin levels after receiving an oral dose of 1800 mg/kg-day for five days (Zvelzer and Apt, 1949). Mice fed 5.3 to 133 mg/kg-day for 90 days did not experience adverse effects (Shopp et al. 1984).

Oral exposure of pregnant rabbits to naphthalene at dosages up to 400 mg/kg/day, using methylcellulose as the vehicle, resulted in no apparent adverse reproductive effects (PRI 1986). When administered in corn oil to pregnant mice, however, a dosage of 300 mg/kg/day resulted in a decrease in the number of live pups per litter (Plasterer et al. 1985).

No information on the toxicological effects of acetone avian receptors was located.

D4-3-1.70 Nickel (CAS No. 7440-02-0)

Small amounts of nickel can be essential for normal growth and reproduction (ATSDR 1988a). Oral exposure to high concentrations of nickel has been reported to adversely affect the hematological system and reproduction.

Rats fed 5 mg/kg/day nickel sulfate in a 2-year dietary study did not produce hepatic changes or altered body weights (Ambrose et al. 1976). This NOAEL was supported by a rat subchronic drinking water study conducted by American Biogenics Corp. (ABC 1986) and a rat reproductive study by Research Triangle Institute (RTI 1987). For mammalian herbivores, a subchronic study of cows that did not exhibit reduced food intake or growth rate when fed 250 mg/kg/d nickel carbonate (O'Dell et al. 1979 as cited in NAS 1980). A dietary study exposing dogs to 1,000 ppm nickel did not result in adverse effects (Ambrose et al. 1976).

In a three-generation study by Ambrose et al. (1976) no adverse effects on fertility, gestation, viability and lactation were noted in rats maintained on diets containing nickel sulfate hexahydrate at 0, 250, 500, or 1,000 ppm nickel.

A study by Eastin and O'Shea (1981) fed mallard ducks nickel at concentrations of: 0, 12.5, 50, 200, or 800 ppm. The ingestion had no effect on egg production, hatchability, or survival of ducklings.

D4-3-1.71 Nitrate (CAS No. 14797-55-8)

Homo sapiens have been identified as the most sensitive species. Several studies (Bosch et al. 1950; Walton 1951; Sattelmacher 1962; Simon et al. 1964) indicate that infants ingestion of formulas made with nitrate-contaminated groundwater at concentrations greater than 10 mg/L caused cyanosis. In infants, the pH of the gastrointestinal system is higher than in adults and this allows for the growth of nitrate-reducing bacteria. These bacteria convert nitrate to nitrite which then causes methemoglobinemia. Therefore, for humans, the NOAEL is 1.6 mg nitrate as nitrogen/kg-day. Nitrates are a normal component of the human and animal diet.

However, in animal studies, the NOAELs and LOAELs identified are typically much higher. In animal studies, Hugot et al. (1980) identified a LOAEL of 900 mg nitrates as nitrogen/kg-day. This LOAEL is based on a three generation study of rats at doses of 90 to 160 mg nitrate as nitrogen/kg-day administered as sodium nitrate. There were no effects on the reproductive capabilities, but small decreases in birth weight, growth rate during lactation, and changes in organ weights at weaning were observed. A LOAEL of 90 mg nitrates as nitrogen was identified, and assuming that 10% of the nitrate is converted to nitrate a LOAEL of 900 mg nitrates as nitrogen/kg-day.

Reproductive NOAELs have been observed for hamsters and mice at 66 mg/kg-day (FDA 1972a,b) when administered on days 6-10 and 6-15 of gestation, respectively. For rabbits and rats, the reproductive NOAEL of 41 mg nitrates as nitrogen/kg-d when administered on days 6-6-18 and 6-15 of gestation, respectively. Another reproductive NOAEL was determined by Sleight and Atallah (1968). This study was for 143 to 204 days for guinea pigs. Four dose levels was administered at 12, 102, 507, and 1130 mg nitrates as nitrogen/kg/-day. Nitrate at the highest dose level reduced the number of live births, but no adverse effects were observed a the other dose levels.

In drinking water, Druckrey et al. (1963) supplied rats with 20 mg nitrates as nitrogen/kg-day for three generations. No teratogenic effects or adverse effects on reproduction were detected in any generation. Assuming that 10% of the nitrate is converted to nitrite, a NOAEL of 200 mg nitrates as nitrogen/kg-day was established.

D4-3-1.72 Nitric acid (CAS No. 7697-37-2)

No information in the literature was found for the toxicity of nitric acid.

D4-3-1.73 Phenanthrene (CAS No. 85-01-8)

Five studies of cancer-initiating activity in skin painting assays in mice have yielded one positive result. Groups of 30 female CD-1 mice received a single dermal application of 1.8 mg phenanthrene in benzene, followed by twice-weekly applications of tetradecanoylphorbol acetate (TPA, 3 mg), a promoter, for 35 weeks (Scribner, 1973). Phenanthrene used in the study was purified by preparative thin-layer chromatography (TLC) and determined to be homogeneous on TLC. It is stated in the report that the dose of TPA was 3 mg (5 umol); however, it is not clear whether this refers to the twice weekly or total dose. Controls were treated with TPA (6 mg); it is not clear whether controls received benzene (vehicle). The tumor incidence (skin papilloma) at 35 weeks was 12/30 (40%) in treated mice and 0/30 in TPA controls. Tumor-initiating activity was not shown in the four other mouse skin painting studies.

Parenterally administered phenanthrene was not shown to have tumorigenic activity in three studies. In the first (Buening et al. 1979), groups of Swiss Webster BLU:Ha ICR mice (100/group, approximately 50% of each sex) received intraperitoneal injections of phenanthrene (total dose 0.25 mg) in dimethyl sulfoxide (DMSO) or DMSO alone on days 1, 8, and 15 after birth. Phenanthrene was > 98% pure and homogeneous on HPLC. Incidence of pulmonary tumors (adenomas) at 38 to 42 weeks was 1/18 (6%) and 5/17 (30%) in female and male treated mice and 7/38 (18%) and 2/10 (19%) in female and male controls. No hepatic tumors occurred in treated or control mice. One treated female mouse developed malignant lymphoma. In the second study (Grant and Roe, 1963), albino mice (sex, strain and group size not specified) received single subcutaneous injections of phenanthrene (40 ug, purity not specified) in an acetone/gelatin vehicle or only the vehicle. Incidence of pulmonary adenomas after 52 to 62 weeks was 3/39 (6%) in treated mice and 8/34 (24%) in vehicle controls. Other tumors reported were 4 hepatomas and 2 skin papillomas in treated mice, and 1 mammary adenocarcinoma, 1 hepatoma and 1 hemangioma in control mice. Finally in the Steiner (1955) study, groups of 40 to 50 male and female C57BL mice (numbers per sex not specified) received single subcutaneous injections of 5 mg phenanthrene (purity not specified) in tricaprylin. No tumors were reported in 27 surviving mice after 4 months (IRIS 1994).

D4-3-1.74 Phenol (CAS No. 108-95-2)

The NTP (National Toxicology Program, 1983) studied developmental effects of phenol in timed-pregnant CD rats. Phenol was administered by gavage at 0, 30, 60, and 120 mg/kg/day in distilled water on gestational days 6 to 15. Females were weighed daily during treatment and observed for clinical signs of toxicity. A total of 20 to 22 females/group were confirmed to be pregnant at sacrifice on gestational day 20. Detailed teratological evaluations were conducted at sacrifice. Results of this study did not show any dose-related signs of maternal toxicity or any clinical symptoms of toxicity related to phenol treatment. The number of implantation sites per litter was approximately the same in all groups, as was the number of live fetuses per litter. However, since implantations in this strain take place prior to gestational day 6 (prior to dosing), no relationships between treatment and number of implantation sites can be established. The most important finding, however, was a highly significant reduction in fetal body weights in the high-dose group. The highest fetal NOAEL in this study was 60 mg/kg/day.

In NCI (1980) rat and mice 90-day subchronic studies, 10 animals/sex/group were exposed to 0, 100, 300, 1,000, 3,000, or 10,000 ppm phenol in water. Decreased water intake and body weight gain were noted for both sexes of rats and mice and rats exposed to the high dose (780 mg/kg/day for rats and 1700 mg/kg/day for mice). Lower doses of phenol exposure did not cause any adverse effects in either rats or mice (234 and 510 mg/kg/day, respectively). The LOAEL for this study was 10,000 ppm.

In a subchronic oral study prepared by Dow Chemical in 1945, 10 rats/group were gavaged 5 days/week with 0, 50, or 100 mg/kg (0, 35.7 or 71.4 mg/kg/day) phenol until 135 or 136 doses were administered. Rats in the high-dose group showed a more marked drop in body weight gain than did other groups, but the group rapidly recovered. Rats in both dosage groups showed some degree of unspecific kidney damage yielding a LOAEL of 50 mg/kg, or 5,000 ppm, for this study.

In a chronic drinking water study conducted by NCI (1980), rats (F344) and mice (B6C3F1) were dosed with 0, 2,500, and 5,000 ppm phenol (rats: 0, 153, 344 mg/kg/day; mice: 0, 313, 500 mg/kg/day) in the drinking water for 103 weeks. All the animals were sacrificed 2 weeks after dosing ceased; detailed histopathological and carcinogenic evaluations of target organs were conducted. Results of this bioassay indicated a dose-related depression in mean body weight gain in both sexes of mice and rats. Animals exposed to both dose levels of phenol showed a significant drop in water consumption (water consumption in mice was severely depressed) resulting in significant body weight depression in the high-dose animals. This study also reported an increased incidence of chronic kidney inflammation in all dosed female rats and in the 5,000-ppm male rats.

A 1938 study reported normal growth and reproduction at phenol concentrations up to 5,000 mg/L (400 mg/kg/day) in a multigenerational rat reproduction study.

In a mouse developmental toxicity study (NTP, 1983) phenol was administered by gavage at 0, 70, 140, or 280 mg/kg/day on gestational days 6 to 15. At the highest dose, 4/36 mice died; no deaths occurred in any other groups. Average maternal body weight gain and weight gain in survivors also were significantly reduced at the highest dose; significant clinical signs of toxicity (tremors) also were seen at that dose level. As in the rat study, there was a highly significant dose-related for reduced fetal body weight, statistically different from controls at the highest dose level. The highest NOAEL in this study was 140 mg/kg/day (IRIS 1994).

D4-3-1.75 Polycyclic Aromatic Hydrocarbons (PAHs)

In general, unsubstituted PAHs do not tend to accumulate in mammalian adipose tissues despite their high lipid solubility (Eisler 1987b). This is probably because PAHs are rapidly and extensively metabolized. Numerous PAHs are distinct in their ability to produce tumors in most mammal species tested. Acute and chronic exposure to various carcinogenic PAHs has resulted in destruction of the hematopoietic and lymphoid tissues, ototoxicity, respiratory epithelia, and other effects (Eisler 1987b). For the most part, tissue damage occurs at dose levels expected to cause cancer; therefore, the threat of malignancy is the predominant health effect of concern. Target organs affected by PAHs are diverse, probably because of the widespread distribution of PAHs in the body and selective attack by PAHs on proliferating cells. Laboratory studies with mice show that many PAHs affect annuals immune systems. Although ecotoxicological data are scarce, the tendency is for many PAHs to be either carcinogenic (high molecular weight compounds) or acutely toxic (low molecular weight compounds) to many organisms. In addition, chronic toxicities, mainly seen as increased frequencies of hyperplasia and neoplasia in aquatic invertebrates, fish, and amphibians, have been demonstrated in areas with high sediment PAH concentration (Eisler, 1987b).

Studies done on mallards revealed no signs of mortality or toxicity during exposure in the adults but produced significant reduction in embryonic growth and a significant increase in the percent of abnormalities, e.g., incomplete skeletal ossification, eye defects, brain, liver, feathers, and bill (Hoffman and Gay 1981).

D4-3-1.76 Polychlorinated Biphenyls (PCBs) (CAS No. 1336-36-3)

PCBs comprise a physicochemically and toxicologically diverse group of 209 compounds whose widespread use and chemical stability make of them ubiquitous in the environment. Because of their generally low acute toxicity, effects on environmental receptors are more likely to be sublethal and chronic than acute. Toxicity and risk assessment of PCB mixtures is complicated by the fact that the 209 congeners differ markedly in both the severity and the nature of their biological effects. The toxic potency of individual congeners is dependent upon their structure. While the approximate isostereomers of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—i.e., coplanar molecules with chlorine atoms in the lateral (but not ortho) ring positions—are the most toxic (and carcinogenic in some species), many others manifest very low acute or chronic toxicity.

The most toxic congeners are also the most potent inducers of mixed-function oxidases as well as some Phase II enzyme activities (reviewed by Safe 1992). These enzymes metabolize not only the inducing PCBs but also a variety of endogenous molecules, such as steroid hormones, that are necessary for normal physiological function. As a result, PCBs may exert adverse effects on development and reproduction in various vertebrate species, including birds (e.g., Koval et al. 1987). In addition, there is considerable difference in the sensitivity of various species to these compounds. Particularly sensitive species include some birds, guinea pigs, and mink (McConnell 1985).

Dahlgren and Linder (1971) and Dahlgren et al. (1972) examined the effects of Aroclor-1254 exposure in pheasants. Although no NOAEL was identified in this work, its focus on a wild species and dosing of both sexes makes it attractive for TRV development. Nine to 10 mg/kg-day Aroclor-1254 reduced sperm concentrations in American kestrels, Falco sparverius (Bird et al. 1983).

Linder et al. (1974) identified NOAELs for Aroclor-1254 in a two-generation reproductive study in rats. Many studies have focused on the toxicity of various PCBs to mink, which is a sensitive species (Eisler 1986b; EPA 1993b). A study by Aulerich and Ringer (1977) revealed that mink are very sensitive to these compounds and that the lethal dose varied inversely with the chlorine content of the PCBs.

In laboratory studies PCBs have been shown to decrease the survivability and hatchability of birds (Schwetz et al. 1974). Porphyria cutanea tarda which is one of the symptoms of embryo mortality, endema, and deformities have been seen in piscivorous birds due to PCB exposure (Gilbertson et al. 1991).

D4-3-1.77 Potassium chloride (CAS No. 7447-40-7)

Very little information in the literature was found for the toxicity of potassium chloride. Potassium chloride has been shown to cause severe ulceration in the gastrointestinal tract at a level equivalent to 1000 mg potassium ion when given to monkeys. Excessive amounts of potassium in the diets of rats can cause an increase in the insulin activity and growth depression (Venugopal and Luckey, 1978).

D4-3-1.78 Potassium hydroxide (CAS No. 1310-58-3)

Potassium hydroxide is toxic by ingestion and inhalation. It is a strong caustic, corrosive to tissue. The TLV for potassium hydroxide is reported as 2 mg/m^3 of air. No other information for the toxicity of potassium hydroxide was found.

D4-3-1.79 Potassium nitrate (CAS No. 7757-79-1)

No information in the literature was found for the toxicity of potassium nitrate.

D4-3-1.80 Potassium phosphate (CAS No. 7778-53-2)

No information in the literature was found for the toxicity of potassium phosphate.

D4-3-1.81 Potassium sulfate (CAS No. 7778-80-5)

No information in the literature was found for the toxicity of potassium sulfate.

D4-3-1.82 Pyrene (CAS No. 129-00-0)

Pyrene can accumulate from soil into roots and translocate them into other vegetation parts (Eisler, 1987). In general, unsubstituted PAHs do not tend to accumulate in mammalian adipose tissues despite their high lipid solubility (Eisler, 1987). This is probably a result of the fact that PAHs are rapidly and extensively metabolized.

Male and female CD-1 mice (20/sex/group) were gavaged with 0, 75, 125, or 250 mg/kg/day pyrene in corn oil for 13 weeks. The toxicological parameters examined in this study included body weight changes, food consumption, mortality, clinical pathological evaluations of major organs and tissues, and hematology and serum chemistry. Nephropathy, characterized by the presence of multiple foci of renal tubular regeneration, often accompanied by interstitial lymphocytic infiltrates and/or foci of interstitial fibrosis, was present in 4, 1, 1, and 9 male mice in the control, low-, medium-, and high-dose groups, respectively. Similar lesions were seen in 2, 3, 7, and 10 female mice in the 0, 75, 125, and 250 mg/kg treatment groups. The kidney lesions were described as minimal or mild in all dose groups. Relative and absolute kidney weights were reduced in the two higher dosage groups. Based on the results of this study, the low dose (75 mg/kg/day) was considered the NOAEL and 125 mg/kg/day the LOAEL for nephropathy and decreased kidney weights.

Groups of 14-29 newborn male and 18-49 newborn female CD-1 mice on 1, 8, and 15 days of age received intraperitoneal injections of pyrene (purity unknown) in dimethyl sulfoxide (DMSO) (total dose = 40, 141 or 466 ug/mouse), or DMSO alone (Wislocki et al. 1986). Tumors were evaluated in animals that died spontaneously after weaning and in all remaining animals at 1 year after exposure. The mid-dose group was initiated 10 weeks after the other groups and had a separate vehicle control. The survival rate in the high-dose groups (male and female) was 25 to 35%; most of the mice died between the last injection and weaning. This high mortality was not observed in the control, low- or mid-dose groups (the survival rates were not stated). A statistically significant increase in the incidence of liver carcinomas occurred in the mid-dose males (3/25) relative to their vehicle control group (0/45), but not in the high-dose males (1/14) or low-dose males (0/29) or in female mice, when compared with their respective controls. The incidences of total liver tumors (adenomas and carcinomas), lung tumors or malignant lymphomas were not statistically significantly elevated in treated animals (IRIS 1994).

D4-3-1.83 Selenium (CAS No. 7782-49-2)

Selenium is a critical nutrient and a key component of several enzymes (Eisler, 1985b). It is often found in high concentrations in areas where soils have been derived from Cretaceous rocks (Eisler

1985b). Selenium does accumulate to high concentrations in certain species of plants (e.g., Aster, Astragalus) (Eisler 1985b). Livestock species ingesting these plants have been reported to exhibit toxic symptoms such as abnormal movements, labored breathing, dilated pupils, bloating, diarrhea, and rapid pulse. No effective treatment is known for counteracting the toxic effects of high levels of ingested selenium. Prolonged exposure to more moderate levels of selenium result in skin lesions involving alopecia, hoof necrosis and loss, emaciation and increased serum transaminases, and alkaline phosphatase in animals (TOXNET 1994). Selenium has been reported to cause growth retardation, decreased fertility, embryotoxicity, fetotoxicity, and teratogenic effects in animals (TOXNET 1994). Birds appear to be particularly susceptible to selenium, particularly in the area of reproductive success. Malformations in chickens and waterfowl have been widely reported (EPA 1993a).

Selenium deficiency is often a greater threat to health than selenium poisoning (Eisler 1985b). Selenium deficiency has been documented in a variety of species including fish, quail, ducks, poultry, rats dogs, domestic grazing animals, antelope, monkeys, and humans (Eisler 1985b). Selenium can also reduce the toxicity of other heavy metals such as thallium, arsenic, and copper (Wilber 1980).

In a study by Rosenfeld and Beath (1954), selenium administered as potassium selenate to sires and pregnant rats through five breeding cycles did not affect reproduction, the number of young raised, or on the reproduction of two successive generations of dams and sires in groups receiving 1.5 ppm selenium. Selenium doses of as low as 3.2 mg/kg body weight have resulted in death in sheep (Eisler 1985b).

D4-3-1.84 Silver (CAS No. 7440-22-4)

The precious metal silver is relatively rare in the earth's crust and does not occur regularly in animal tissue. As a result, the toxicity of silver has been little studied. Approximately 1 to 10% of ingested silver is absorbed; as much as 18% may be retained. The extent of silver absorption is an inverse function of gastrointestinal transit time (Furchner et al., 1968). One week after dosing with 0.0011 to 0.000005 mg/kg silver, the dose retention was less than 1% in rodents, with a high transit time, and approximately 10% in dogs, which have a low transit time. First pass metabolism further reduces silver bioavailability. Silver-protein complexes accumulate in the liver, and biliary excretion (complexed with glutathione) is the major route of elimination. In most tissues, silver is deposited as large granules. With rare exceptions, these deposits are not associated with adverse effects. The LD_{50} of silver in rats is relatively high at 24 mg/kg (reviewed by Rungby 1990).

Silver causes a conditioned deficiency of selenium in rats, decreasing tissue levels of selenium, and the selenoprotein glutathione peroxidase (Ganther 1980). Silver ions complex strongly to sulfhydryl groups and cause preoccupation of hepatocellular membrane lipids (Rungby et al. 1987; Shinogi and Maeizumi 1993). Because of its affinity for sulfhydryls, the degree of binding to cellular macromolecules and toxicity of silver is mitigated by inducing the divalent metal-binding protein metallothionein (Shinogi and Maeizumi 1993). Exposing fetal and adult rats to silver results in deposition in the central nervous system (Rungby and Danscher 1983a, b). Pyramidal cells in the developing hippocampus appear to be a sensitive target, exhibiting reduced cellular volume in both pre- and postnatally exposed rate (Rungby et al. 1987; Rungby 1990).

A study by Rungby and Danscher, (1984) in which mice exposed to approximately 18 mg/kg/day for 4 months were observed to be "hypoactive." Although silver deposits occurred in certain motor centers of the brain, no association between the concentration of deposits and the extent of hypoactivity was found. Neither the reproductive nor the developmental toxicity of this contaminant has been adequately studied.

No information on the toxicological effects of silver on avian receptors was located.

D4-3-1.85 Sodium (CAS No. 7440-66-6)

Sodium salts are generally considered to be nontoxic, although some anions contribute greatly to the toxicity of such salts as sodium chromate. Arsenate, and vanadate. The hydroxyl ions contribute to the caustic action of the alkali metal hydroxides (Venugopal and Luckey, 1978). Sodium is essential for all living organisms to maintain osmotic pressure, body fluid balance, and hydration of the tissues.

Sodium toxicity is best assessed by using NaCl. Excessive intake of NaCl in mammals causes a tendency to drink large amounts of water, diarrhea caused by osmotic changes in the digestive tract, stiff gait, salivation, muscular fibrillation, exhaustion, and death. The pathological signs at autopsy include violent local inflammatory reaction in the gastrointestinal tract caused by capillary and venous congestion of the lamina propia and submucosa of the pyloric stomach, small bowel, cecum, and colon, and dehydration in the other body organs such as the adrenals, brain, liver, and spleen. Death is attributed to respiratory failure associated with acute encephalopathy and congestion of body organs. Chronic toxicity symptoms include inhibition of growth, increased water intake and urinary volume, and osteosclerosis. Sublethal doses cause nausea, vomiting, abdominal stress, convulsive seizures, and diarrhea (Venugopal and Luckey, 1978).

A chrinic oral dose (LD100 in 100 days) in albino rats was shown to be 2.06-3.23 g NaCl/kg per 24 hr. Only one acute toxicity study had available data for sodium. An LD_{50} was reported for mice given 4,000 mg/kg intraperitoneally (TOXNET).

No data on the toxicological effects of sodium to avian receptors were available. No other information was found for the toxicity of sodium.

D4-3-1.86 Strontium (CAS No. 7440-24-6)

Strontium is moderately toxic by ingestion and inhalation. The stable form has low toxicity. Sr compounds are chemically and biologically similar to calcium. The oxides and hydroxides are moderately caustic materials (Sax and Lewis 1987). The toxic effect of excessive Sr intakes is inhibition of calcification of epiphyseal cartilage and deformities of long bones at high doses. Because strontium is a metabolic analog of calcium, strontium is readily absorbed from the lung, gastrointestinal tract, or blood stream (dermal exposure). The strontium that is retained in the body, in a large part, is deposited in the bone (Driver 1994).

Storey (1961) fed young and adult female rats (3/group) diets with adequate calcium, phosphorous, and vitamin D for 20 days. The dietary levels of Sr (as Sr carbonate) given both adult and young rats correspond to 190, 380, 750, 1,000, 1,500, and 3,000 mg/kg-day for young rats and 95, 190, 375, 750, and 1,500 mg/kg-day for adult rats. Rats were examined for changes in bone mineralization and defects in cartilage. Young rats were found to be affected more severely at lower dietary strontinum levels than were adult rats. For young rats, the dietary level of 190 mg/kg-day was an NOAEL and 350 mg/kg-day was an LOAEL. In adults, the first obvious bone change occurred at the 750 mg/kg-day dietary Sr level. For adult rats, the dietary level of 375 mg/kg-day was an NOAEL and 750 mg/kg-day was an LOAEL.

Marie et al. (1985) administered stable Sr to weanling male Spraque-Dawley rats. Rats were divided into groups (8/group) receiving 0, 316, 425, 525, and 633 mg/kg-day. Rats in the 633 mg/kg-day dose group showed signs of increased mineralization lag time; excessive osteoid thickness associated with

a decline in the rate of calcification, which resulted in slow growth rate; and defective long bone growth. This study identified an NOAEL of 525 mg/kg and an LOAEL of 633 mg/kg-day.

Skoryna (1981) investigated the oral toxicity of stable Sr male adult RVH hooded rats. The rats were administered experimental doses of Sr chloride corresponding to 70, 147, and 263 mg/kg-day Sr. The control and experimental groups received adequate amounts of calcium and magnesium in their drinking water. Except for bone, no organ predilection for Sr was observed. A chronic NOAEL of 263 mg/kg-day was identified from this study (IRIS 1994).

No information on the toxicological effects of Sr on avian receptors was located.

D4-3-1.87 Sulfate (CAS No. 14808-79-8)

Sulfates are generally of low toxicity. Several studies indicate no adverse effects when sulfate compounds are administered (Brown and Gamatero 1970; Sasse and Baker 1974; Paterson et al. 1979) and others that list the effects of loose feces and decrease intake (Bird 1972; L'Estrange et al. 1969). These five studies were conducted using pigs, chicken, and sheep. One study listed an LD_{50} for a single-dose injection of sodium sulfate monohydrate in mice of 45.6 mg/kg/d (Nofre et al. 1963).

No other information was found for the toxicity of sulfate.

D4-3-1.88 Sulfuric Acid (CAS No. 7664-93-9)

Sulfuric acid is a colorless to cloudy liquid. Fuming sulfuric acid (oleum) has a sharp, penetrating odor. Concentrated sulfuric acid is extremely irritating, corrosive, and destructive to all living matter not by virtue of its acidity but because of its affinity for water. The affinity is so strong that it will remove the elements of water from even anhydrous organic matter such as carbohydrates, resulting in charring or carbonization with the liberation of heat. The fumes of oleum are initially composed of sulfur trioxide which will combine with water, either present in the air or on the mucous membranes of exposed organisms to form sulfuric acid. Effectively then, exposure to sulfur trioxide is equivalent to exposure to sulfuric acid. The respiratory tract is the main target area affected by sulfuric acid fumes.

Amdur et al. (1952) found the 8-hour LC_{50} of sulfuric acid aerosol of mass diameter of 1 um to be 18 mg/cu for 1 to 2 month old guinea pigs and 50 mg/cu for 18 month old animals. The cause of death in the animals dying within 2 hours appeared to be asphyxia caused by bronchoconstriction and laryngeal spasm.

A study conducted by Cavender et al. (1978) using a mist of sulfuric acid in combination with ozone on guinea pigs and rats reported no direct effects on the respiratory system due to the sulfuric acid.

D4-3-1.89 Terphenyl (CAS No. 26140-60-3)

There are three isomeric terphenyls: ortho, meta, and para. Terphenyls attack the skin, respiratory system, and eyes, causing eye and skin irritation and thermal burns (Sittig, 1981).

In 30 day feeding studies, rats fed 250 and 500 mg/kg-day of ortho-terphenyl had elevated liver and kidney weight ratios. Animals fed the same amount of meta-terphenyl showed only elevated kidney weight ratios. These effects were not observed in animals fed para-terphenyl. Results of histopathological studies on these animals were similar to controls (ACGIH, 1980). Insignificant weight

decreases and intensification of antitoxid functions of liver were observed in rats orally administered 2.5 to 5.0 g/kg-day for 1 month (Patty, 1981-82).

No information on the toxicological effects of terphenyls on avian receptors was located. No other information was found for the toxicity of terphenyl.

D4-3-1.90 Tetrachloroethene (PCE) (CAS No. 127-18-4)

PCE is an apparent liver carcinogen in mice. Animals exposed to PCE exhibit CNS depression as well as cardiovascular, liver, and kidney effects when exposed to short, high doses of PCE via inhalation (Rowe et al. 1952). Eleven consecutive daily oral doses of 100 mg PCE/kg BW resulted in liver changes in mice (Schuman et al. 1980).

Oral LD_{50}s of 8,100 mg/kg (mice) and 8,850 (rat) have been reported (RTECHS Database, 1984; TOXLINE Database, 1984). Little data are available on effects associated with long-term, chronic exposure to PCE.

Buben and O'Flaherty (1985) exposed mice to PCE in corn oil gavage at doses of 0, 20, 100, 200, 500, 1,500, and 2,000 mg/kg 5 days per week for 6 weeks. These doses correspond to a daily dose of 0, 14.3, 71, 142, 355, 1,065, and 1,420 mg/kg-day. Increased liver triglycerides were first observed in mice treated with 100 mg/kg. Liver weight/body weight ratios were significantly higher than controls for animal treated with 100 mg/kg. At higher doses, hepatotoxid effects were observed. Adjusting 20 mg/kg for the treatment schedule of 5 days/week, a NOAEL of 14 mg/kg-day was established.

The symptoms of acute intoxication from this material are the result of its effect upon the nervous system. It is an experimental carcinogen (Sax and Lewis, 1987).

No information on the toxicological effects of tetrachloroethylene on avian receptors was located.

D4-3-1.91 Thallium (CAS No. 7440-28-0)

Thallium is a nonvolatile heavy metal element that is not used extensively by industry, but is mainly introduced into the environment as a waste product of other metals. Thallium can exist in the atmosphere as an oxide, a hydrazide, a sulfate, or a sulfide. Thallium is present in mono- or trivalent forms in the environment. Thallium(III) forms some organometallic compounds and thallium (1) forms relatively few complexes with the exception of those with halogen, oxygen, and sulfur ligands. Thallium can be removed from solution by adsorption onto clay minerals, bioaccumulation, or (in reducing environments) precipitation of the sulfide. Increased pH values have been found to produce extensive thallium-humic acid interactions while lowering thallium-inorganic interactions. Thallium may be bioconcentrated by living organisms (Callahan et al. 1979). Thallium(I) is more stable and resembles the alkali metal cations in many of its chemical properties. Thallium(III) forms many organic compounds (Zitko 1975), the toxicity of which has been little explored.

Thallium is slightly more acutely toxic to mammals than mercury. The similarity between kinetic profiles of inorganic trivalent and monovalent thallium species suggests that they are converted in vivo to one chemical form, probably monovalent thallium (Sabbioni et al. 1980). Isomorphic with potassium, thallium (I) is readily absorbed and distributed throughout the body, and can substitute for potassium and other monovalent cations in enzymatic reactions. The affinity of thallium (I) for enzymes is 10 times higher than that of potassium, which may cause the observed toxic effects (Zitko 1975). Thallium (I)

uncouples oxidative phosphorylation, adversely affects protein synthesis, and inhibits a number of enzymes including alkaline phosphatase and succinic dehydrogenase (Zitko 1975). Thallium is also toxic to plants, inhibiting chlorophyll formation and seed germination.

A study in the 1930s of the acute toxicity of thallium sulfate in game birds including quail (Shaw 1933) formed the basis for the TRV for these functional groups. In a study of the acute toxicity of thallium sulfate in three immature golden eagles (Aquila chrysaetos), the acute oral LD_{50} was estimated to be between 60 and 120 mg/kg (Bean and Hudson 1976). Using the lower end of this range as the QCE, a TRV for raptorial birds at the INEL was derived.

Rats exposed to thallium in their drinking water have shown effects on various neurological (Manzo et al. 1983. Rossi et al. 1988) and reproductive (Formigli et al. 1986) endpoints. Because of the clear ecological relevance of reproductive impairment, a QCE was selected from the study of thallium-induced testicular toxicity (Formigli et al. 1986).

D4-3-1.92 Tin (CAS No. 7440-31-5)

The toxicity of tin differs dramatically depending on whether it is in an inorganic or organic form. Inorganic tins are not highly toxic to poor absorption and rapid turnover rate in tissues (Eisler 1989). Inorganic tin has 10 stable isotopes with compounds existing in either the 2+ (stannous) or 4+ (stannic) oxidation state. Bioconcentration of organotin compounds has been reported in aquatic environments. However, the ability of higher organisms to reduce organotins into forms that can be readily excreted seems to preclude food chain biomagnification (Eisler 1989).

Signs of inorganic tin toxicity in mammals include vomiting, diarrhea, and eye and nose irritation (Eisler 1989). Systemic effects can include ataxia, paralysis, and growth retardation. At extremely high doses, testicular degeneration, pancreatic atrophy, and kidney necrosis can occur (Eisler 1989). Monoorganotin compounds have a generally low toxicity. Effects of organotins can include neuromuscular effects and liver toxicity. In general, triorganotin compounds are the most toxic group. However, large inter- and intraspecies differences exist in resistance to organotin toxicity.

TRVs were generated for mammalian receptors using the results of two studies: a 4-week feeding study exposing rats to stannous sulfate by deGroot et al. (1973), and a study by Kimbrough (1976) involving a single oral dose of inorganic tin in dogs.

The chemical, physical, and biological properties of inorganic tin compounds differ dramatically from those of representative organotin compounds (Eisler 1989). Inorganic tins are poorly absorbed, rapidly turned over to tissues and are essential for growth in the rat. Organotins have been shown to cause intrahepatic and extrahepatic choloangitis in rats at high dietary levels.

No toxicity information on inorganic tin exposure in birds was located.

D4-3-1.93 Toluene (CAS No. 108-88-3)

Most of the toxicological data for toluene focus on exposures via inhalation. EPA (1984b) reported that pregnant mice treated with 430 to 870 mg/kg-day by gavage experienced an increased fetal mortality rate. Maternal toxicity was not observed.

The oral toxicity of toluene was evaluated in a subchronic gavage study (NTP 1989). Male and female rats were given toluene in corn oil at levels of 0, 223, 444, 888, 1,775, or 3,550 mg/kg-day for 13 weeks. Groups of 10 rats per sex per treatment group were used. All animals receiving 3,550 mg/kg-day died within the first week. Toxic effects including prostration, hypoactivity, ataxia, piloerection, lacrimation, excessive salivation, and body tremors were observed in animals given the 1,775 mg/kg-day dose. Pathological findings include changes in liver, kidney, and bladder weights. Histopathologic liver lesions, hepatocellular hypertrophy, and nephrosis were observed in animals given 1,775 mg/kg-day. No toxic effects were seen in animals receiving 888 mg/kg-day or less (IRIS 1994).

No information on the toxicological effects of toluene on avian receptors was located.

D4-3-1.94 Total Petroleum Hydrocarbons (TPH)

Petroleum is a combination of several products in varying amounts and combinations. Petroleum is composed of but is not limited to: Gasoline, Diesel, Fuel Oil No.2, Fuel Oil No.4, Kerosene, JP-4, JP-5, and Used Oil. Each of these products is a complex mixture of several hundred hydrocarbon compounds (PAHs, benzene, toluene, ethylbenzene, xylenes, ethylene dibromide, 1,2-dichloroethane, and methyl tert-butyl ether) and other additives (e.g., anti-knock agents, corrosion inhibitors, anti-oxidants, etc.). The actual composition of these products varies depending on the source, age, temperature, and other factors and conditions. Thus, no unique composition exists for any of the aforementioned products. The behavior of these products in the environment and their toxic effects depends on the properties of the individual constituents and their concentrations (State of Idaho 1996).

Although no toxicological data are available for TPHs per se, data were obtained for JP-4, a jet fuel petroleum product. No studies on the teratogenicity, embryotoxicity, or reproductive effects are available. Although no LD_{50} was found for JP-4, and oral LD_{50} of 20 g/kg has been reported for kerosene in guinea pigs. Chronic inhalation studies have been conducted with JP-4 in rats, mice, and dogs. No other information was found for the toxicity of TPHs.

The TRVs for benzene was used for TPHs and is thought to have similar toxicity properties.

D4-3-1.95 Tributyl phosphate (CAS No. 126-73-8)

Limited information is available on the health effects of tributyl phosphate. One study (Smyth 1944) indicates that the LD_{50} for tributyl phosphate is 3,000 mg/kg-d. Patty (1963) indicates that large doses of tributyl phosphate administered to rats either orally or intraperitoneally causes damage to the central nervous system, weakness, dyspnea, twitching, and pulmonary edema. No other information was found for the toxicity of tributyl phosphate.

D4-3-1.96 Trichloroethylene (CAS No. 79-01-6)

Trichloroethene is a central nervous system depressant, and causes liver and kidney pathology. Studies report that this compound may bioaccumulate with continuous exposure, due to the fact that only one-third of the administered dose is excreted in the urine as metabolites in the 24-hour period following exposure.

Tucker et al. (1982) found that mice survived 14 daily gavage doses of trichloroethylene at 240 mg/kg. When trichloroethylene was given in drinking water for 6 months, mice survived dosing at

the highest levels (660 mg/kg/day, males, and 793 mg/kg/day, females). An oral LD_{50} for trichloroethylene was reported at 2,402 mg/kg for male mice.

A study by the NTP (1986) on F344 rats exposed to concentrations of trichloroethylene as high as 300 mg/kg/day experienced a decrease in the number of live litters per breeding pair. The same study concluded that there was no effect on the fertility of the F0 and F1 generations but did state that the effects were probably due to the toxicity of the trichloroethylene.

Significant increases in the incidence of liver tumors have been reported in B6C3F1 mice of both sexes. Malignant lymphomas and pulmonary adenocarcinomas were also reported in mice.

D4-3-1.97 Trimethylolpropane-triester (CAS No. 15625-89-5)

Little data on the toxicity of trimethylolpropane-triester are available. Only one toxicity study was identified in the available literature for trimethylolpropane-triester. An oral LD_{50} of 5,190 L/kg was reported for rats (Toxicol. Appl. Pharm., 1974; TOXNET).

No data on the toxicological effects of trimethylolpropane-triester to avian species were available. No other information was found for the toxicity of trimethylopropane-triester.

D4-3-1.98 Uranium-234, 235, 238 (CAS No. 7440-61-1)

Natural uranium contains three isotopes U-234, U-235 and U-238 (U-230 occurs in nature but has a short half-life of 20.8 days). The percent abundance of each isotope in natural uranium is, respectively, 0.006%, 0.72% and 99.27% (ATSDR 1990b). Uranium can be found in the earth's crust at an average concentration of 2 ppm.

In natural uranium, the radioactivity from U-238 accounts for about half the total radioactivity, and the radiation from U-234 and U-235 accounts for the other half. Uranium emits primarily alpha particles that are unable to penetrate skin, but can travel short distances in the body if they are inhaled or ingested. Natural uranium emits very small amounts of gamma rays that can penetrate the skin, so there is little, if any, danger from this type of radiation from uranium (ATSDR 1990b). Moreover, no animal studies have definitively linked inhalation or oral exposure to natural uranium to development of cancer. Based on the specific activity of the uranium isotopes, lists the oral SF for U-234, U-235, and U-238 as 1.60E-11/pCi, with an inhalation SF of 2.60E-08/pCi. For U-238 the inhalation SF is 2.40E-08/pCi (EPA 1994b).

With regard to noncancer health risks associated with uranium, exposure to natural concentrations of uranium in food, water, air and soil does not appear to have any toxic effects. Animals that have had oral, inhalation, or dermal exposure to large amounts of uranium have developed damage to the kidney tubules, but other systems were not affected.

Overall, studies in animals and humans also indicate that exposure to uranium is unlikely to produce immunological or neurological effects. Although the data are conflicting, animal studies indicate that exposure to uranium may affect fetal weight and skeletal development in animals, and may possibly alter the ratio of male to female live births in areas where people have excessive exposure to uranium (ATSDR 1990b).

D4-3-1.99 Vanadium (CAS No. 7440-62-2)

Vanadium occurs naturally in igneous rock, and shales, in some uranium and iron ores and in association with fossil fuels. In the environment, vanadium is usually combined with oxygen, sodium, sulfur, or chloride (ATSDR 1990). There is no indication that vanadium is nutritionally required by higher plants and annuals (Ammerman et al. 1973). Vanadium uptake into above ground parts of terrestrial plants is low. However, some legumes have been identified as vanadium accumulators (ATSDR 1992c). In general, bioconcentration and biomagnification in terrestrial environments appears limited.

Most toxic effects of vanadium are associated with inhalation of vanadium pentoxide (ATSDR 1992c). Vanadium is poorly absorbed in the gastrointestinal tract and most is excreted unabsorbed in feces (ATSDR 1992c). Ingestion of high levels of vanadium are reported to cause dehydration, emaciation, and diarrhea (Ammerman et al. 1973).

A study of vanadium toxicity in female leghorn chickens by (Kubena and Phillips 1982) was used to develop a TRV of 0.85 mg/kg/day. A TRV of 0.25 mg/kg/day was derived using a study of the effects of vanadium to mallards (White and Dieter 1978).

A study of the effects of vanadium to mice (Schroeder and Balassa 1967) was used to derive a TRV of 0.5 mg/kg-day for vanadium. There is little information in the literature regarding vanadium toxicity in ruminants (Ammerman et al. 1973). A study was used to derive a TRV of 0.42 mg/kg/day (Abbey 1968).

D4-3-1.100 Xylene (CAS No. 1330-20-7)

Acute exposure to xylene via inhalation primarily caused central nervous system effects, although acute liver injury was observed in guinea pigs given 1 to 2 g/kg-day intraperitoneally (WHO, 1981). An oral LD_{50} value of 4300 mg/kg has been reported for rats (1984, TOXNET).

Chronic studies indicate that xylene has a relatively low toxicity over the long-term. No changes were found in rats, guinea pigs, dogs, and monkeys continuously exposed to 80 ppm for 127 days nor in rats exposed to 700 ppm for 130 days (WHO, 1981).

Ungvary et al. (1980) evaluated the toxicity of xylene in rats. Rats were exposed via inhalation to 35, 300, or 700 ppm continuously on days 7 through 14 of gestation. No adverse effects were observed, and the authors concluded that xylene was not teratogenic.

A commercial mixture of xylene was given to mice via gavage at doses of 0; 520; 1,030; 2,060; 2,580; 3,100; or 4,130 mg/kg-day on days 6 through 15 of gestation (Marks et al. 1982). No adverse effects were observed in either dams or fetuses exposed to levels of 1030 mg/kg-day or less. An exposure of 2,060 mg/kg-day and higher approached lethal levels in dams. Fetal weight was significantly decreased and the average percentage of malformations in fetuses significantly increased at these dose levels.

Groups of 50 male and 50 female Fischer 344 rats and 50 male and 50 female B6C3F1 mice were given gavage doses of 0, 250, or 500 mg/kg/day (rats) and 0, 500, or 1000 mg/kg/day (mice) for 5 days/week for 103 weeks. The animals were observed for clinical signs of toxicity, body weight gain, and mortality. All animals that died or were killed at sacrifice were given gross necropsy and comprehensive

histologic examinations. There was a dose-related increased mortality in male rats, and the increase was significantly greater in the high-dose group compared with controls. Although increased mortality was observed at 250 mg/kg/day, the increase was not significant. Although many of the early deaths were caused by gavage error, NTP (1986) did not rule out the possibility that the rats were resisting gavage dosing because of the behavioral effects of xylene. Mice given the high dose exhibited hyperactivity, a manifestation of CNS toxicity. There were no compound-related histopathologic lesions in any of the treated rats or mice. A NOAEL of 250 mg/kg-day was developed.

No data on the toxicological effects of xylene to avian receptors were available.

D4-3-1.101 Zinc (CAS No. 7440-66-6)

Zinc is found naturally in the environment and is present in all foods (ATSDR 1988b). It is an essential element and occurs in the environment in the 2+ state. Zinc is likely to be strongly sorbed to soil. Relatively little land disposed zinc is expected to be in a soluble form. Bioconcentration factors of soil zinc by terrestrial plants, invertebrates, and mammals are 0.4, 8, and 6, respectively (ATSDR 1988b).

Excessive dietary zinc has been shown to cause copper deficiency and anemia (ATSDR 1988b). Cadmium has also resulted in the redistribution of zinc to the liver and kidney. Health effects associated with zinc exposure include anemia, liver necrosis, fetal resorption, and in extreme cases, cessation of reproduction (ATSDR 1988b).

A study by Drinker et al. (1927) conducted on dogs and cats fed 175 to 1000 mgm over the course of 3 to 15 weeks observed no damage from the daily ingestion of zinc oxide. The concluded that most of the zinc was removed in the urine and feces of the animals.

A study of sheep by Allen et al. (1983) revealed pathological changes in liver and kidney.

High mortality rates were reported in mallard ducks fed zinc. Reduction in pancreas and gonads were also observed. The reduction in the gonads was so significant that the ability to reproduce was probably lost. Partial paralysis, diarrhea, and weight loss were noted in the first 10 days of treatment (Gasway 1972).

D4-3-1.102 Zirconium (CAS No. 7440-67-7)

There is little information on the toxicity of zirconium. No studies on the developmental or reproductive effects are available and there are a limited number of studies available on the adverse effects of zirconium to animals. Schroeder et al. (1968 and 1970) administered 0 and 5 ppm zirconium sulfate to both male and female Long-Evans rats and CD mice for their lifetime. No adverse effects on survival, growth, or tumor incidence were noted in rats and a slight decrease relative to the controls of survival. Nonsignificant changes in the glucose levels were noted in the rats, but this may have been related to a chromium deficiency.

A study by Harrison et al. (1951) used groups of 10 male and female rats for dose levels of 0, 0.2, 2.0, or 20% by weight of a moist paste of zirconium carbonate containing 20.9% zirconium dioxide. No significant differences from controls were seen for any of these endpoints at any dose levels and no rats died. This highest dose level of 1.12 mg/kg-day represents a NOAEL.

D4-3-2. REFERENCES

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Appendix D4
Attachment 4

Fate and Transport

Appendix D4
Attachment 4

Fate And Transport of COPC

The following section discusses the environmental fate and transport for contaminants identified as COPCs for ecological receptors at the INEEL. Environmental fate properties are important because they provide information on the environmental behavior of contaminant compounds throughout the various environmental media.

INORGANICS

Aluminum

Aluminum is a naturally occuring element in the earth's crust that is always found in combination with other elements or ores (ATSDR). Aluminum comprises approximately 8% of the earth's crust, making it the third most abundant element (Brusewitz 1984). Aluminum metals, aluminum compounds and materials have a wide variety of uses (Sax and Lewis 1987). Aluminum metal is primarily used as a structural material in the construction industry. Aluminum is used extensively in the production of metal alloys and in the electrical industry. Aluminum powder is used in paints and protective coatings. Natural aluminum minerals, such as bentonite and zeolite, are used in water purification.

The major anthropogenic source of aluminum-containing particulate matter is from coal combustion (Lee and Von Lehmden 1973). Anthropogenic sources account for about 13% of atmospheric aluminum. The largest source of particle-borne aluminum is the flux of dust from ores and rock materials in the earth's surface. A significant amount of dust is attributed to volcanic activity.

Aluminim may be considered immobile, as evidenced by the formation of bauxite, which is an accumulated product of weathering. However, there is data to suggest that aluminum does become mobilized in low pH (<4) soils in areas prone to acid rain (Peterle, 1991). The element of aluminum is considered nondegradable in the environment.

The fate of aluminum in surface water is complex and variable. Aluminum forms many soluble complexes with chloride, fluoride, nitrate, and sulfate ions, as well as fulvic and humic acids (USEPA, 1988).

Aluminum exists in many forms in soil. In soils with pH less than 5.0, exchangeable aluminum is found in the trivalent form. In an alkaline medium, aluminum is present as $(Al)OH_4$ (USEPA, 1983). Aluminum is highly unstable in the normal pH ranges of soils and readily oxidizes to aluminum (3+) (Lindsay, 1979). Only small quantities of soluble aluminum were found in soils that had pH values between 4.7 and 7.8 (Gough et al., 1979).

Aluminum is abundant in most soils and makes up 2 to 12 percent (20,000 to 120,000 mg/kg) of soils (Jackson, 1964). In general, decreasing pH results in an increase in mobility for monomeric forms of aluminum (Goenaga and Williams 1988), which is of concern with respect to the occurrence of acid rain and the release of acid mine drainage. Only small quantities of soluble aluminum were found in soils that had pH values between 4.7 and 7.8 (Gough et al, 1979).

The mobility of aluminum in sediments is dependent upon the solubility of the aluminum compound and upon the pH of the medium. Specific information was not found on the bioavailability of aluminum in sediments. However, it is expected to be similar to bioavailability in soils.

Aluminum chloride

No information in the literature was found for the fate and transport of aluminum chloride.

Aluminum hydroxide

No information in the literature was found for the fate and transport of aluminum hydroxide.

Aluminum nitrate

No information in the literature was found for the fate and transport of aluminum nitrate.

Aluminum nitrate nonahydrate

No information in the literature was found for the fate and transport of aluminum nitrate nonahydrate.

Aluminum sulfate

No information in the literature was found for the fate and transport of aluminum sulfate.

Ammonia

Ammonia is both a natural and a manmade chemical. The major source of ammonia in the world is thought to be soil. Small amounts of ammonia are produced industrially as a by product of the coking of coal. The largest proportion of industrial ammonia production occurs in areas where natural gas is cheap and plentiful. Ammonia and ammonium compounds used as fertilizer represent 80% of the commercially produced ammonia, with fiber and plastics, explosives, and other uses accounting for 10, 5, and 5% respectively (C & E News, 1987). The small proportion of commercially produced ammonia not incorporated into fertilizers is used as refrigerant, a corrosion inhibitor, in the purification of water supplies, and as a component of household cleaners. Ammonia is used to manufacture of pharmaceuticals and explosives, and in the production of various chemical intermediates (LeBlanc et al. 1978; Sax and Lewis 1987).

Atmospheric ammonia can be readily removed from the air by rain or snow washout (Adamowicz 1979; Kumar 1985). Ammonia is the predominant basic gas in the atmosphere. As such, it is capable of rapidly reacting with gaseous H_2SO_4, HNO_3, and HCl, forming ammonium aerosols which can then undergo dry deposition (Irwin and Williams 1988).

If released to surface water, ammonia volatilizes to the atmosphere. The rate of volatilization of ammonia from water will increase with increasing pH and temperature, and can depend on other environmental factors as well. Adsorption of ammonia to sediment and suspended organic material can be important under proper conditions. Adsorption to sediment should increase with increasing organic content, and decreasing pH. Ammonia, however, can be produced in, and subsequently released from sediment (Jones et al. 1982; Malcolm et al. 1986). Because ammonia, as ammonium ion, is the nutrient of choice for many plants (Rosswall 1981), uptake of soil ammonia by living plants is an important fate

process. The uptake of ammonia is rapid. It is either rapidly taken up by plants, bioconverted by the microbial population, or volatilized to the atmosphere. Because of these processes, ammonia does not leach readily through soil; thus, it is rarely found as a contaminant of groundwater. However, nitrate derived from ammonia may penetrate groundwater.

Antimony

Antimony is found in small amounts in the earth's crust (ATSDR 1992). Antimony is a brittle metal that is not readily fabricated and has no significant use in its unalloyed state. It is alloyed with lead and other metals to increase their hardness, mechanical strength, corrosion resistance, and electrochemical stability or decrease their coefficient of friction. The most common end-use of antimony compounds is antimony trioxide for fire retardation. Antimony trioxide in a suitable organic solvent is used as a fire retardant for plastics, textiles, rubber, adhesives, pigments, and paper (U.S. Bureau of Mines 1989).

Antimony is moderatley mobile in oxidizing environments with pHs between 5.0 and 8.0, and is immobile in reducing environments (Rose et al. 1979). Antimony may exist in the oxidation states –3, 0, +3 and +5, although the existence of simple Sb^{+3} and Sb^{+5} ions is improbable. Under moderately oxidizing conditions, antimony has a valence of +3 and the higher acid, H_3SbO_4, in which the element exhibits a valence of +5, is found only in high oxidizing conditions. Metallic antimony is ordinarily quite stable and is not readily attacked by air or moisture. Under controlled conditions, antimony will react with oxygen to form the oxides Sb_2O_3, SbO_4, and Sb_2O_5.

Antimony is released to the atmosphere in the form of particulate matter or adsorbed to particulate matter. It is dispersed by wind and removed by gravitational settling and dry and wet deposition (Schroeder et al. 1987).

Antimony released into waterways is generally associated with particulate matter; it is transported to and settles out in areas of active sedimentation such as where a river empties into a lake or bay (Beijer and Jernolov 1969). Antimony is usually evenly concentrated and distributed throughout the sediment but does not bind strongly to the sediment.

Antimony may accumulate with heavy elements in carbonaceous shales or become sorbed on clays and hydrous oxides. Antimony may be volatilized when stibine (SbH_3) or its methylated derivatives are formed during the reduction of antimony in soils. Antimony has an affinity for clay and other mineral surfaces. Less than 10% of the antimony in sediments from both contaminated and uncontaminated sediments was bound to easily oxidizable organic matter (Callahan et al. 1979). Since antimony has an anionic character in aqueous solution, it probably has little affinity for complexation with humic and fulvic acids. Little information is currently available on the behavior of antimony in soil; however, current data suggests that it is relatively immobile in any soil type (USPHS, 1991a).

Arsenic

Arsenic is used in wood preservatives, in agricultural chemicals (principally herbicides and desiccants), in glass, in nonferrous alloys, and more recently in semiconductors (U.S. Bureau of Mines 1988).

Arsenic released to the atmosphere as a gas vapor or adsorbed to particulate matter may be transported to other media via wet or dry deposition. Trivalent arsenic may undergo oxidation in the air, and arsenic in the atmosphere is usually a mixture of the trivalent and pentavalent forms (EPA 1984a).

Arsenic may be relatively mobile in oxidizing environments, controlled by coprecipitation with iron oxides, and in iron-poor or partially reducing environments, as suggested by moderately high average content in water relative to rock (Rose et al. 1979).

Arsenic in water can undergo a complex pattern of transformations, including oxidation-reduction reactions, ligand exchange, biotransformation, precipitation, and adsorption (Callahan et al. 1979), making it extremely mobile in aquatic systems. The factors that most strongly influence arsenic mobility in aquatic systems are Eh, pH, metal sulfide and sulfide ion concentrations, iron concentration, presence of phosphorus minerals, temperature, salinity, and distribution and composition of biota (Callahan et al. 1979).

The dominant form of arsenic present in aerobic soils is As^{+5}, while As^{+3} is the primary species in anaerobic soils. Inorganic arsenic is more mobile than organic arsenicals and thus is more likely to leach into surface or groundwaters. Trivalent species are generally more toxic, more soluble, and more mobile and pentavalent forms. Soil microbes can metabolize arsenic to volatile arsine forms. The half-life of arsenic in soil is estimated to be 6.5 years for arsenic trioxide to 16 years for lead arsenate. Arsenates sorb most readily to soils with high organic matter content, low pH, low phosphate, and low mineral content (Eisler 1988a).

Many arsenic compounds are readily solubilized in soil, making them available for plant uptake or for reduction by organism or chemical interactions. Biological uptake of arsenic results in measurable quantities of reduced or methylated arsenic forms. Arsenic occurs naturally in all environmental media. Biotransformation of these compounds may occur and yield volatile arsenicals. The bioavailability of arsenic depends on several factors including pH, soil texture and fertility level, and plant species. In general, arsenic is most available to plants grown in coarse soils having little colloidal material and a low ion-exchange capacity. Conversely, fine soils high in clay, organic matter, iron, calcium, and phosphate tend to retard the bioavailability of arsenic to plants (NRCC 1978). The accumulation of arsenic in plants tends to be directly correlated with the amount of arsenic in the dissolved fraction versus total arsenic concentrations (NRCC 1978).

Asbestos

In the United States, asbestos is mainly mined in open pits. In the past asbestos was blasted or drilled from the pits but an alternative method has been developed to reduce air emissions. This method uses bulldozers and scrapers to remove the ore from the pit. The main uses for asbestos are in paper products, asbestos cement products, friction products, textiles, packing and gaskets, coatings, and asbestos-reinforced plastics.

Asbestos fibers are nonvolatile and insoluble, so their natural tendency is to fall out of air and water and be deposited in soil or sediment (Callahan et al. 1979; Fuller 1977). However, some fibers are sufficiently small that they can remain in suspension in both air and water and be transported long distances. For example, fibers with aerodynamic diameters of 0.1 to 1 um can be carried thousands of kilometers in air (Jaenicke 1980), and transport of fibers over 75 miles has been reported in the water of Lake Superior (Callahan et al. 1979). Adsorptive interactions between the fibers and natural organic contaminants may favor coagulation and precipitation of the fibers (Bales 1985; Callahan et al. 1979).

Asbestos fibers in water may undergo dissolution of some of the metal ion and hydroxyl ion content, but the basic silicate structure of the fiber remains intact (Morgan and Holmes 1986).

Asbestos fibers in soil are not known to undergo significant transformation or degradation.

Barium

Barium is distributed all over the earth and occurs most frequently as barite. Barium is used as a carrier for radium, deoxidizer for copper, lubricant for anode rotors in X-ray tubes, in plants, soap, paper, rubber, in the manufacture of ceramics and glass, and as a heat stabilizer for plastics. Barium metal in the free state does not occur in nature. It is found in zinc or iron ores. It is emitted mostly by industrial processes involved in the mining, refining, and production of barium and barium-based contaminants and as a result of the combustion of coal and oil.

In the atmosphere, barium is likely to be present in particulate form (EPA 1984a). Although chemical reactions may cause changes in speciation of barium in air, the main mechanisms for the removal of barium compounds from the atmosphere are likely to be wet and dry deposition (EPA 1984).

Barium adsorbs to soils through reactions with metal oxides and hydroxides. In reducing environments, barium is moderately mobile, and in oxidizing environments, with pH vlaues between 5.0 and 8.0, it is slightly mobile (Jensen and Bateman 1981). Barium mobility in soil is reduced by the precipitation of barium carbonate and sulfate. Humic and fulvic acid have not been found to increase the mobility of barium (EPA 1984a). Barium is taken up, retained, and excreted in mammals in much the same way as calcium compounds.

Beryllium

Pure beryllium metal is used in X-ray transmission windows, missile parts, nuclear-reactor neutron reflectors, nuclear weapons, fuel containers, precision instruments, rocket propellants, aircraft disc brakes, navigational systems, heat shields, and mirrors (EPA 1987a). Beryllium oxide is used in high-technology ceramics, electronic heat sinks, electrical insulators, microwave-oven components, gyroscopes, military-vehicle armor, rocket nozzles, crucibles, thermocouple tubing, and laser structural components (EPA 1987a).

Removal of beryllium from the atmosphere results from wet and dry deposition (EPA 1987a). Because most atmospheric beryllium results from coal combustion it is likely that the chemical form would be beryllium oxide.

The mobility of beryllium is limited in ore environments by beryl's extreme resistance to weathering. The element beryllium is considered nondegradable in the environment.

Soluble beryllium salts are hydrolyzed to form insoluble beryllium hydroxide, which would have low solubility in the pH range of most natural waters (Callahan et al. 1979). Complexing with hydroxide ions may increase solubility somewhat, but it is likely that in most neutral environments beryllium is present in particulate form rather than the dissolved form.

In most types of soil, beryllium is expected to be tightly adsorbed because it displaces divalent cations which share common sorption sites (Fishbein 1981). Due to its geochemical similarity to aluminum, beryllium may be expected to adsorb onto clay surfaces at low pH and be complexed into some insoluble compounds at high pH (Callahan et al. 1979).

Boron

Boron, which does not occur naturally in its elemental form, typically occurs as sodium borate (borax) or as a calcium borate (colemanite) (Micromedex, 1996). Elemental boron is used in atomic reactors as a neutron absorber (EPA 1986). Boron is also used in cleaners and in antifreeze.

In soil, pH factors and the presence of other metals control the bioavailability of boron. Although boron is adsorbed to an increased extent as soil pH increases above 7 standard units, there is only weak adsorption at lower pH values (Wild, 1988). However, Overcash and Pal (1979) reported that the principal adsorption fate for boron is in association with aluminum oxides. The pH dependence of boron adsorption is such that for soils at pHs from 5.5 to 7 standard units, boron would adsorb to exchangeable aluminum and would become unavailable. In general, boron adsorption will be most significant in soils that contain high concentrations of amorphous aluminum and iron oxides and hydroxides.

Borates are relatively soluble in water, and will probably be removed from the atmosphere by precipitation and dry deposition (EPA 1987). Adsorption-desorption reactions are expected to be the only significant mechanism that will influence the fate of boron in water (Rai et al. 1986).

Cadmium

Cadmium is a silver-white, blue-tinged, lustrous metal. Cadmium oxidizes readily but because it is highly resistant to corrosion it is widely used to plate iron and steel. Cadmium is also used in solders, nickel-cadmium batteries, and in stabilizers for polyvinyl chloride.

Cadmium released to the atmosphere in particulate matter can be transported some distance and transferred to other environmental compartments via wet or dry deposition. Given cadmium's tendency of being concentrated in very small particles, particularly those in fly ash, it is likely to be more persistent in the atmosphere than larger particulate pollutants. The transformation of cadmium compounds in the atmosphere is usually by solubility in water and dilute acids.

It is insoluble in water, although its chloride and sulfate salts are relatively soluble in water. The concentration of cadmium in water is usually inversely related to the pH value and amount of organic material present. Cadmium does not form volatile compounds in the aquatic environment, nor does biological methylation occur.

The availability of cadmium in soils depend upon soil pH, cation exchange capacity, chemical speciation, and many other factors. Adsorption and desorption process tend to influence the concentration of cadmium in natural waters. Adsorption and desorption occur rapidly in mud solids and particles of clay, silica, humic material, and other naturally occurring solids. Cadmium tends to remain in the upper portion of the soil profile. Its bioavailability depends on adsorption/desorption rates, pH, and speciation. Changes in physiochemical conditions, especially pH and redox potential, that occur during dredging and disposal of Cd-polluted sediments may increase chemical mobility and, hence, bioavailability of sediment-bound Cd (Khalid et al. 1981). Cadmium is moderately mobile in normal surface weathering ($pH < 4.0$), slightly mobile near oxidizing sulfide ore bodies (pH 5.0 to 8.0), and immobile in organic-rich environments (Callahan et al. 1979).

Cadmium uptake by plants is influenced by the concentration of calcium, sulfides, and sulfites present in the soil. Calcium and cadmium are considered to have the same uptake site; thus, levels of calcium present in soil could limit the amount of cadmium taken up by plants. Cadmium availability to plants is affected by redox potential and pH. Humus-bound and sorbed cadmium contribute to the plant

available pool. Availability may be reduced by higher organic matter content and higher cation exchange capacity (Eisler 1985a).

Calcium

No information in the literature was found for the fate and transport of calcium.

Cerium chloride

No information in the literature was found for the fate and transport of cerium chloride.

Chloride

No information in the literature was found for the fate and transport of chloride.

Chromium

The metallurgical, refractory, and chemical industries are the fundamental users of chromium. Chromium is used as rust and corrosion inhibitors, in toners for copying machines, in water treatment, and as a catalyst (USDI 1988).Chromium is a multivalent element and can exist in the +2, +3, and +6 oxidation states. The latter two, chromium (III) and chromium (VI), are the most stable in the environment.

Chromium is immobile in normal surface weathering (pH 5.0 to 8.0) and in organic-rich environments because of its incomplete inner electron shells, which cause the element to be strongly adsorbed (Rose et al. 1979). Chromium is present in the atmosphere primarily in particulate form. Atmospheric particulate matter is deposited on land and water via wet and dry deposition.

Since chromium compounds cannot volatilize from water, transport from water to the atmosphere is not likely. Most of the chromium released into water will ultimately be deposited in the sediment. Soluble chromium generally accounts for a small percentage of the total chromium. Chromium primarily exists in the oxidation states Cr (III) and Cr (IV) in aqueous systems. They are believed to be interconvertible under natural conditions. Schroeder and Lee (1975) found that Cr (IV) can be reduced by iron, dissolved sulfides, and certain organic compounds with sulfhydryl groups, while Cr (III) can be oxidized by an excess of manganese dioxide and, at a slower rate, by O_2 under natural water conditions. Cr (IV) is a strong oxidizing agent and reacts with reducing materials to form trivalent chromium.

In soils and sediments, chromium is influenced by oxidation/reduction reactions and can be absorbed on the mineral and organic exchange complex or exist as a coating in iron and manganese hydrons oxides particles. Moreover, chromium can remain in solution in the pore water phase, become chelated by an organic ligand, or precipitated (Adriano 1986; Callahan et al. 1979). The sorption of chromium (IV) by hydrous metals oxides and other soil mineral components decreases as pH increases. The presence of other anions (e.g. sulfate and phosphate) significantly affects the extent of absorption by competing for absorption sites. Formation of ion pairs, such as dissolved calcium chromate, can also reduce the extent of absorption. In contrast to chromium (VI), the sorption of chromium (III) increases with pH. In general, it appears from laboratory studies that chromium (III) is absorbed more strongly than chromium (VI). Organic material may also be an important adsorbent in sediments and soils. Slight enrichment of chromium occurs in the humic fraction. Typically, in normal, well-drained arid soils, the great majority of chromium is in the form of chromium (III).

Cobalt

Cobalt has a wide variety of uses including its use in superalloys (alloys that maintain their strength at high temperatures approaching their melting points) and as a catalyst. The most abundant of the radioactive isotopes of cobalt, Co-60, is produced in nuclear explosions and in reactors. Its radiological half-life is 5.27 years (Eisenbud 1987).

Cobalt is slightly mobile in normal surface weathering (pH 5.0 to 8.0), moderately mobile near an oxidizing sulfide ore body, and immobile in organic-rich environments (Perel'man 1967). The element cobalt is considered nonbiodegradable in the environment.

The transport of atmospheric cobalt depends on its state (e.g. gas, vapor, or particle) and on meteorological conditions such as wind, precipitation, topography, and vegetation. The transport of cobalt from atmosphere to soil and surface water occurs as a result of dry and wet deposition.

As for most metals, sediment and soil are the final repository for cobalt emitted into the environment by humans. Most of the cobalt released into water eventually reaches lakes via the transport river transport of dissolved and suspended particles. Cobalt is not significantly adsorbed by organic materials (e.g. humic and fulvic materials) in water.

The transport of cobalt in soil depends on its adsorption/desorption. Cobalt is retained by oxides such as iron and magnesium oxide, crystalline materials such as aluminosilicate and geothite, and natural organic substances in soil. Cobalt has a tendency to form soluble complexes with dissolved organic matter. In clay soil, the adsorption may be due to ion exchange at the cationic sites on clay with either simple ionic or hydrolized ionic species such as $CoOH^+$. At higher soil pH, the mobility of cobalt decreases, probably due to the formation of hydroxide or carbonate. The distribution coefficient of cobalt in a variety of soils ranges from 0.2 to 3800. Therefore, in most soils, cobalt is more mobile than lead, chromium, zinc, and nickel, but less mobile than cadmium (Baes and Sharp 1983; King 1988; Smith and Carson 1981).

Copper

Copper is one of the most important metals because of its durability, ductility, malleability, and electrical and thermal conductivity. It is used primarily as the metal or in alloys. A small percentage of copper production goes into the manufacture of copper compounds, primarily copper sulfate (used in metal finishing)(Jolley and Edelstein 1987).

In general, copper is very mobile in oxidizing and in acidic waters; moderately mobile near an oxidizing sulfide ore body ($pH < 4.0$), and immobile in organic-rich environments.

Copper is released to the atmosphere in the form of particulate matter or adsorbed to particulate matter. It is removed by gravitational settling (bulk deposition), dry deposition, washout by rain, and rainout (Schroeder et al. 1987). Most copper deposited in soil from the atmosphere, agricultural use, and solid waste and sludge disposal will be strongly absorbed and remain in the upper few centimeters of soil.

Copper is one of the least mobile of the trace elements and tends to be uniformly distributed in the soil horizon. Persistence in soils is due to binding to organic matter, its formation of oxides with iron and manganese, the presence of clay minerals, and soil pH. A pH of 6 or less increases the mobility and availability of copper in soil. Copper is one of the trace elements most extensively complexed by humic materials. Most copper is readily available to plants when soil pH is below 6, especially in soils with low

organic matter and humic material content. Sulfides, which are abundant in soils under reducing conditions, effectively precipitate copper. Biogenic ligands bind with copper, resulting in the precipitation and sorption of copper. Copper in soil tends to strongly bind with organic matter, which limits its availability for uptake by plants. Soil parameters that appear to influence copper availability include pH, CEC, and organic matter content.

Much of the copper discharged into waterways is in particulate matter and settles out, precipitates out, or adsorbs to organic matter, hydrous iron and manganese oxides, and clay in sediment or in the water column. Copper in soil tends to strongly bind with organic matter, which limits its availability for uptake by plants. Copper has a pronounced tendency to form complexes with both organic and inorganic ligands. Soil parameters that appear to influence copper availability include pH, CEC, and organic matter content.

No evidence of bioaccumulation was obtained from a study of pollutant concentrations in the muscle and livers of 10 mammal species (Hernandez et al. 1985). No evidence of copper biomagnification in the food chain was observed.

Fluoride

In addition to occurring naturally, fluorides are used as insecticides; in the manufacture of aluminum, steel, glass, cement, bricks, high octane gasoline, and phosphate fertilizers; and for water treatment (McKee and Wolf, 1963).

Vaporization and aerosol formation are the most important processes involved in partitioning of fluorides between surface waters and the atmosphere. Atmospheric fluorides are transported to soils and surface waters though wet and dry deposition processed (NAS 1971).

Fluorides in surface waters that are not volatilized to the atmosphere can partition to sediments or biota (NAS 1971). Precipitation of insoluble fluorides from surface waters is dependent on the amount of calcium for complexing (Drury et al. 1980). Only a small amount of the fluoride released to the soil every year is leached to the groundwater.

In soils with high calcium or aluminum content, fluorides form persistent complexes. In sandy, acidic soil, water soluble forms predominate. Soluble fluoride compounds are almost completely fixed in soils as calcium fluoride at pH 6.5 or above if sufficient calcium carbonate is available (Brewer 1966).

Hydrofluoric acid

No information in the literature was found for the fate and transport of hydrofluoric acid.

Iron

Important environmental fate and transport mechanisms include chemical transformation/degradation through oxidation/reduction reactions and hydrolysis. The mobility of iron is dependent on the element's valence state. Fe^{2+} is moderately mobile, whereas Fe^{3+} has a very low tendency to mobilize because it is precipitated as hydrous iron oxides with pH > 2.0 (Hem 1960).

Solubility properties of iron indicate that exposing an equilibrated system to relatively small shifts in Eh or pH can cause great changes in iron solubility. Thus, when pyrite is exposed to oxygenated water or ferric hydroxide is in contact with reducing substances, iron will tend to go into solution. Chemical

speciation, which is affected by pH, Eh, and oxygen content in aquatic systems, further affects the concentration and availability in the environment.

Organic compounds containing iron are particularly important in life processes, such as photosynthesis, and in the functions of hemoglobin in the blood of animals. As an essential nutrient, iron is accumulated by plants and animals; although no literature was available, it seems reasonable to assume that the majority of accumulation results from uptake rather than dermal absorption.

Lead

Lead is a ubiquitous trace constituent in rocks, soils, plants, water, and air. Lead is neither essential nor beneficial to living organisms. Lead is a bluish-gray, soft metal. Lead is used abundantly in the storage battery industry. Substantial amounts are also used in gasoline additives, pigments, ceramics, pesticides, and plumbing (Paone, 1970).

Lead is slightly mobile in normal surface weathering conditions (pH 5.0 to 8.0) and immobile in organic-rich reducing environments (Perel'man 1967). The relatively low mobility of lead is attributed to the element's tendency to absorb to manganese-iron oxides and insoluble organic matter. In the presence of moisture, an oxide film forms on lead, which, with carbon dioxide, forms a white carbonate. The oxidized solid, PbO_2, is stable under highly oxidizing conditions. Under reducing conditions in the presence of sulfur, lead combines to form lead sulfate.

Metallic lead is relatively insoluble in hard waters. Lead has four stable isotopes: Pb-204 (1.5%), Pb-206 (23.6%), Pb-207 (22.6%), and Pb-208 (52.3%). Lead occurs in four valence states: elemental (Pb^0), monovalent (Pb^+), divalent (Pb^{+2}), and tetravalent (Pb^{+4}). In nature, lead occurs mainly as Pb^{+2} and is oxidized to Pb^{+4}. Some lead salts are somewhat soluble in water. Of the organoleads, tetraethyllead and tetramethyllead are most stable and highly soluble in many organic solvents but fairly insoluble in water. Both undergo photochemical degradation in the atmosphere to elemental lead and free organic radicals. Organolead compounds are primarily anthropogenically-produced (Eisler 1988b).

In water Pb is most soluble and bioavailable under conditions of low pH, low organic content, low concentrations of suspended sediments, and low concentrations of the salts of calcium, iron, magnesium, zinc, and cadmium. Lead and its compounds tend to concentrate in the water surface microlayer (the upper 0.3 mm), especially when surface organic materials are present in thin films (Demayo et al. 1982).

Most Pb entering natural waters is precipitated to the sediment bed as carbonates or hydroxides (May and McKinney 1981). Lead is readily precipitated by many common anions; desorption and replacement by other cations is extremely slow (Boggess 1977).

In the sediments, Pb is mobilized and released when the pH decreases suddenly or ionic composition changes (Demayo et al. 1982). Some lead may become methylated when temperatures increase, the pH is reduced, and by microbial activity.

Magnesium

No information in the literature was found for the fate and transport of magnesium or magnesium fluoride.

Manganese

Metallic manganese (ferromanganese) is used principally in steel production to improve hardness, stiffness and strength. Manganese dioxide is commonly used in the production of dry-cell batteries (EPA 1984).

The common, naturally occurring oxides for manganese are generally forms on MnO_2, when the manganese is at the Mn^{4+} oxidation state, or mixed-valence oxides in which the oxidation state of manganese is less than +4 but substantially above +3. Manganese in all oxidation states is easily hydrolyzed (Cotton and Wilkinson 1972). The precipitation of these hydrous manganese oxides is considered to be one of the principal mechanisms for the control of heavy metals concentration in soils and sediments (Hem 1964).

The transport and partitioning of manganese is influenced by solubility of the particular form present, which, in turn, is determined primarily by pH oxidation/reduction potential. Manganese-containing particles are mainly removed from the atmosphere by gravitational settling (EPA 1984). Manganese may exist in one of four oxidation states: 2+, 3+, 4+, and 7+. Divalent manganese (Mn^{+2}) exists mostly in waters with a pH of 4-7. Manganese is often transported in rivers as suspended sediments. Manganese in water may be significantly bioconcentrated at lower trophic levels but biomagnification of manganese in the food chain does not appear to be significant (EPA 1984).

The likelihood that soluble manganese compounds will sorb to soils is affected primarily by the cation exchange capacity and organic matter content of the soil. Soil sorption can vary by as much as five orders of magnitude depending on soil conditions. The oxidation state of manganese in soil may be altered by microbial populations (ATSDR 1990). In general, the lower the pH, the less sorption into the soils. Therefore, once manganese is sorbed in the soils or sediments, it will remain there unless it is taken up into biological systems.

Mercury

The most important industrial uses for mercury involve electrical apparatus, chloralkai production, water-base paint, and agricultural fungicides.

Mercury is moderately mobile in normal surface weathering conditions (pH 5.0 to 8.0) and near an oxidizing sulfide ore body (pH < 4.0). It is immobile in organic-rich, reducing environments (Perel'man 1967).

Mercury exists in the environment in three oxidation states: the element itself, +1 (mercurous) state, and +2 (mercuric) state. The factors that affect which species will dominate in the environment are the redox potential and the pH of the system. In a moderately oxidizing environment above pH 5.0, the predominant mercury species will be elemental mercury. Particle-bound mercury can be converted to insoluble mercury sulfide, which can be bioconverted into more soluble or volatile forms that may re-enter the atmosphere or be taken up by biota and bioaccumulated in the terrestrial food chain. Mercury forms many stable organic complexes that generally are more soluble in organic matter than in water. Mercury strongly absorbs to inorganic and organic particles. Mercury can be transformed in the environment by biotic and abiotic oxidation and reduction, bioconversion of organic and inorganic forms, and photolysis. Mercury can be strongly concentrated by living organisms (Callahan et al. 1979).

In the aquatic environment, under naturally occurring conditions of pH and temperature, Hg may become methylated by biological or chemical processes or both (EPA 1980a). Methylmercury is the most

hazardous mercury species due to its high stability, its lipid solubility, and its possession of ionic properties that lead to a high ability to penetrate membranes in living organisms (Beijer and Jernelov 1979). All mercury discharged into rivers, bays, or estuaries as elemental mercury, inorganic divalent mercury, phenylmercury, or alkoxyalkyl mercury can be converted into methylmercury compounds by natural processes (Jernelov 1969). The mercury methylation in ecosystems depends on mercury loadings, microbial activity, nutrient content, pH and redox condition, suspended sediment load, sedimentation rates, and other variables (NAS 1978).

Molybdenum

Shales and granite are the major rocks contributing molybdenum to soils (USEPA, 1983). Molybdenum is soluble at high pH values. At low pH (2 to 4.5), molybdenum is strongly sorbed to soil colloids and organic matter, although organic matter is not a major means of rendering molybdenum unavailable (USEPA, 1983). At soil pH values above 5, molybdenum generally is found as the molybdate anion, MoO_4^{2-}) and is transported to the leaves, where it accumulates (USEPA, 1983). Manganese decreases molybdenum solubility by holding molybdenum in an insoluble form. No other information was found for the fate and transport of molybdenum.

Nickel

Nickel is used in stainless and heat-resistant steels, electroplating, batteries, and fuel cells, in chemical production, and as an industrial catalyst (Chamberlain 1987; Tien and Howson 1981).

Nickel is continuously transferred between, air, water, and soil via various natural processes including weathering, runoff, erosion, and leaching. Nickel is very persistent in both soil and water. In the atmosphere, nickel exists primarily in the aerosol form (Schmidt and Andren 1980).

Nickel is slightly mobile in normal surface weathering conditions (pH 5.0 to 8.0), moderately mobile near an oxidizing sulfide ore body (pH 4.0), and immobile in organic-rich reducing environments. Nickel is almost always found in the divalent oxidation state in aquatic systems (Cotton and Wilkinson 1972). Under aerobic conditions and pH below 9.0, nickel forms compounds with hydroxide, carbonate, sulfate, and naturally occurring organic ligands that are sufficiently soluble to maintain aqueous Ni^{2+} concentrations above 10^{-6} M (Callahan et al. 1979). Hydrolysis of aqueous nickel to hydroxide, $Ni(OH)_2$, is significant only under basic conditions. Above pH 9.0, precipitation of the hydroxide or carbonate occurs and exerts some control over nickel's mobility.

Nickel can exist in water in various soluble and insoluble forms depending upon the physicochemical properties of the water. The mobility of nickel in aqueous media is affected by complexation, precipitation/dissolution, oxidation/reduction, and absorption/desorption.

The average residence time of nickel in soil is estimated to be 2,400 to 3,500 years (Nriagu 1980, Grandjean 1984). Although nickel is very persistent in soil, it can leach into groundwater. The sorption of nickel to soils correlates with pH, total iron, and organic matter content. Organic complexing agents in soil tend to restrict the movement of nickel by forming organo-nickel compounds. Nickel is fairly mobile in soils in a low pH and cation exchange capacity (ATSDR 1988a).

Nitrate

The fate of nitrates in the environment is linked to the nitrogen cycle. The nitrogen cycle is a complex biochemical process in which various forms of nitrogen are altered by nitrogen fixation,

assimilation, and reduction of nitrate to N_2 by denitrification (Wetzel 1983). Denitrification by bacteria is the biochemical reduction of nitrate, with concomitant oxidation of organic matter (Wetzel 1983). In addition, all inorganic nitrates act as oxygen carriers, and practically all nitrates are powerful oxidizing agents (Sax and Lewis 1989).

With regard to nitrates, the nitrogen cycle can be viewed as atmospheric deposition (both wet and dry) of nitrates to soils and surface waters. This involves either leaching of nitrates from soils to groundwater or water erosion of nitrates from soils to surfacewater and, finally, returning of nitrogen compounds, including nitrates, to the atmosphere as particulate matter (Roy F. Weston, Co. 1987).

In nonpolluted areas, atmospheric nitrate is the result of oxidation of atmospheric ammonia, much of which originates from the decomposition of terrestrial organic matter (Hutchinson 1944; 1975). In polluted areas, atmospheric nitrates are often in the form of particulate matter carried by the wind. Precipitation and dry fallout transport atmospheric nitrates to the ground and to surface waters (Wetzel 1983).

In general, nitrate will move with water through soil pore spaces (Gustafson 1983). Key factors that influence nitrate migration from soil to surface water and groundwater are soil type and rainfall. For example, clay soil tends to retain nitrates twice as much as sandy soil (Roy F. Weston, Co. 1987). Gustafson (1983) also found that nitrates adsorbed or adhered to soil particles during dry periods were leached through the soil to the aquifer during the spring runoff, decreasing surface nitrate.

Nitrate in the common form of inorganic nitrogen entering fresh waters from precipitation, surface waters, and groundwater. Nitrates in aquatic systems are assimilated into organic nitrogenous compounds within organisms. The biochemical reduction of nitrate and concomitant oxidation of organic matter is described by Wetzel (1983) as bacterial denitrification. During normal metabolism of these organisms, and at death, nitrogen is released as ammonia, which is the primary nitrogenous end product of decomposition of proteins and other nitrogenous organic compounds (Wetzel 1983). According to Wetzel, denitrification occurs in anaerobic environments such as eutrophic lakes. The rate of denitrification also is subject to many environmental factors, including water temperature and pH (Keeney 1973; Wetzel 1983).

Sediments are an important reservoir of nitrogen and the site of much nitrogen metabolism. Denitrification rates in lake sediments are three to four orders of magnitude greater than those of the overlying water (Wetzel 1983); however, denitrification activity within the sediments is related to the reducing conditions (Jones 1979). In sediments of the littoral zone or those in contact with oxygenated water, denitrifying activity is depressed at the sediment surface and increases at sediment depths where reducing conditions increase. As overlying water becomes anoxic and reducing, increasing denitrifying activity occurs at the sediment-water interface (Wetzel 1983). Therefore, nitrate degradation is fastest in anaerobic conditions (HSDB 1991).

Nitric acid

No information in the literature was found for the fate and transport of nitric acid.

Nitrite

No information in the literature was found for the fate and transport of nitrite.

Potassium

No information in the literature was found for the fate and transport of potassium.

Potassium chloride

No information in the literature was found for the fate and transport of potassium chloride.

Potassium hydroxide

Potassium hydroxide (CASRN 1310-58-3), also known as caustic potash, potassium hydrate, or lye, is a white, deliquescent substance with crystalline fractures that absorbs water and carbon dioxide from the air. Potassium hydroxide is soluble in water. It is produced from the electrolysis of concentrated potassium chloride solution. It is used in soap manufacture, bleaching, fuel cells, absorbent for carbon dioxide and hydrogen sulfide, dyestuffs, liquid fertilizers, herbicides, paint removers, and other products. No other information was found for the fate and transport of potassium hydroxide.

Potassium nitrate

No information in the literature was found for the fate and transport of potassium nitrate.

Potassium phosphate

No information in the literature was found for the fate and transport of potassium phosphate.

Potassium sulfate

No information in the literature was found for the fate and transport of potassium sulfate.

Selenium

Selenium is distributed widely in nature and is found in most rocks and soils at concentrations between 0.1 and 2.0 mg selenium/kg (Fishbein 1983). Elemental selenium is seldom found naturally. Selenium is obtained primarily as a byproduct of copper refining (Fishbein 1983). Selenium is used in several products and processes like photoelectric cells, blasting caps, stainless steel, dehydrogenation-catalyst, photoconductors, and in lubricants.

Selenium is moderately mobile in normal weathering conditions (pH 5.0 to 8.0) and in acid and alkiline environments (Perel'man 1967). In aerobic waters, selenium is present in the selenite or selenate oxidation state of +4 or +6. Sodium selenate is one of the most mobile selenium species due to its high solubility and inability to adsorb to soil particles. Hydrogen selenide is highly reactive in air and is rapidly oxidized to elemental selenium and water (NAS 1976), but the other compounds can persist in air. Selenium is stable in four valence states: -2, 0, +4, and +6. The positive anions are present in oxyanoins and organo-selenium compounds. The inorganic forms dominate the physical chemistry of selenium in solution. Inorganic selenium is extremely insoluble and will sorb strongly to soil. Selenium can be absorbed by iron and manganese hydroxides, organic matter, and iron sulfides. Selenium can become mobile in soil/water systems under alkaline conditions. Chemical speciation control solubility. Under low pH conditions, more insoluble elemental selenium tends to be present (Callahan et al.1979). Thus, most of the selenium in aquatic systems is probably transported as the dissolved species.

The volatile selenium compounds that partition into the atmosphere include the inorganic compounds selenium dioxide and hydrogen selenide and the organic compounds dimethyl selenide and dimethyl diselenide. Selenium compounds released to the atmosphere can be removed from atmosphere by dry or wet deposition to soils and surface water.

Silver

Silver is used in a wide variety of products and processes including photographic material, industrial-alloys, electrical and electronic products, electrical contacts, silver paints, and batteries.

Silver is a transitional metal with one stable cationic state, Ag^{+}. Chemical speciation, sorption, and bioaccumulation are important factors affecting the fate and transport of silver. Silver in the atmosphere is released as an aerosol and is deposited with precipitation. In water, the major forms of silver are as the monovalent ion in the form of sulfate, bicarbonate, or sulfate salts; as part or more complex ions with chlorides and sulfates; and as an integral part of, or adsorbed onto, particulate matter (Boyle 1968). Callahan et al. (1979) stated that sorption is the dominant process leading to the partitioning of silver in sediments. Silver is moderately mobile in normal weather conditions (pH 5.0 to 8.0) and near an oxidizing sulfide ore body (pH <4.0); it is immobile in organic-rich, reducing environments (Perel'man 1967).

The mobility of silver in soils is affected by drainage (silver tends to be removed from well-drained soils); oxidation-reduction potential and pH conditions (which determine the presence of iron and manganese complexes which tend to immobilize silver); and the presence of organic matter (which complexes with silver and reduces its mobility) (Boyle 1968). In the absence of organic matter, silver tends to be removed from soils.

Ionic silver is found in aqueous systems as the univalent species, although it can form compounds in the $Ag(2^{+})$ or $Ag(3^{+})$ states (Cotton and Wilkenson, 1972). Metallic silver is stable over much of the Eh-pH range of water, and formation of the metal (which has very low solubility) may exert a control on its mobility. Oxidation/reduction potential has an indirect effect on silver speciation in that it dictates the behavior of sulfur and manganese species, which are important controls on silver. In aquatic environments, it has been suggested that the silver species complexes with chloride, bromide, and iodide ions which control the amount of free silver present, subject to the effects of physical parameters and other anions and cations.

Numerous plants and primary consumer organisms accumulate silver, apparently primarily as a function of sorption/desorption from sediments (Callahan et al. 1979).

Sodium

Sodium is a metallic element that, in its pure form, is soft, silver-white solid that oxidizes rapidly in air. Sodium decomposes water on contact with the evolution of hydrogen to form sodium hydroxide. It is derived from the electrolysis of fused mixtures of sodium chloride and calcium chloride. It is a severe fire risk if in contact with water in any form, ignites spontaneously in dry air when heated, and forms a strong caustic irritant to tissue. No other information was found for the fate and transport of sodium.

Sodium chloride

No information in the literature was found for the fate and transport of sodium chloride.

Sodium hydroxide

No information in the literature was found for the fate and transport of sodium hydroxide.

Sodium nitrate

No information in the literature was found for the fate and transport of sodium nitrate.

Sodium phosphate

No information in the literature was found for the fate and transport of sodium phosphate.

Sodium sulfate

No information in the literature was found for the fate and transport of sodium sulfate.

Strontium

Strontium (Sr) is an alkaline earth element and is, therefore, chemically similar to calcium and barium. Strontium follows calcium through food chains from environmental to organism, but some degree of discrimination exists against strontium (Kirchman et al. 1993). Both strontium and calcium are retained in vertebrate organism primarily in bone. No other information was found for the fate and transport of strontium.

Sulfate

No information in the literature was found for the fate and transport of sulfate.

Thallium

Thallium compounds have a variety of uses. Thallium sulfate is used in the semiconductor industry and in low range thermometers, optical systems, and photoelectric cells, and as a chemical intermediate for other thallium compounds and thallium metals (HSDB 1989).Thallium is a non-volatile heavy metal element that is not used extensively by industry and is mainly introduced into the environment as a waste product of other metals.

Adsorption and bioaccumulation are the most significant fate processes; however oxidation influences the speciation and solubility. No information was found specifically on the rate of oxidation of thallium in aquatic environments; but in very oxidizing water, the most oxidized form of thallium, Tl(III), amy be present (Callahan et al. 1979). Thallium exists in water primarily as a monovalent ion, but may be trivalent in highly oxidizing water (Callahan et al. 1979). The monovalent form can complex with other minerals and form highly soluble species such as thallium chloride, sulfate, carbonate bromide and hydroxide (USPHS 1990).

Thallium can exist in the atmosphere as an oxide, a hydrazide, sulfate, or sulfide. These thallium compounds are not volatile (EPA 1983). Thallium sulfate and thallium hydroxide will partition into water vapor so precipitation may remove these forms from the atmosphere (EPA 1988). Thallium oxides are less soluble in water, and may be subject to only atmospheric dispersion and gravitational settling.

Thallium has valences of +1 and +3. Thallium (III) forms some organo-metallic compounds and thallium (I) forms relatively few complexes with the exception of those with halogen, oxygen, and sulfur ligands. Thallium can be removed from solution by absorption onto clay minerals, bioaccumulation, or (in reducing environments) precipitation of the sulfide. Thallium may partition from water to soils and sediments. Increased pH values have been found to produce extensive thallium-humic acid interaction while lowering thallium-inorganic interactions (Callahan et al. 1979).

Tin

The principal use of tin is in the making of containers (HSDB 1989), including aerosol cans and miscellaneous food and beverage containers (WHO 1980). Tin is used to coat copper wire, and as a soldering material. Alloys of tin are used to make nuclear reactor components (tin-zirconium) (WHO 1980).

Tin may be transported in the atmosphere by the release of particulate matter derived from the combustion of fossil fuels and solid wastes. Tin in aerosol samples that exist in particulate-carbon masses is removed from the atmosphere predominantly by gravitational settling (Byrd and Andreae 1986).

The solubilities of organotin compounds in water are not well known. At ambient temperatures, their solubilities range from 10 ug/L to about 50 mg/L (Laughlin and Linden 1985; WHO 1980).

Tin in water may partition to soils and sediments. Cations such as Sn^{+2} and Sn^{+4} will generally be adsorbed by soils to some extent, which reduces their motality. Tin is generally regarded as being relatively immobile in the environment (WHO 1980).

Uranium

Uranium (U) is found in all rocks and soils. Acid igneous rocks (high silica content) contain a concentration of the order of 3 ppm, about 100 times greater than ultra-basic igneous rocks but considerably less than the phosphate rocks of Florida and southeastern Idaho and neighboring areas. The phosphate rocks in these areas contain as much as 120 ppm. The high U content of phosphate rocks is reflected in high U concentrations in commercial phosphate fertilizers. The uranium normally found in nature consists of four isotopes: U-230, U-234, U-235, and U-238. U-238 is present in the amount of 99.28%, has a half-life of 4.5×10^9 years and a specific activity of 3.3×10^5 pCi/g, and is usually in equilibrium with U-234 (0.0058%). U-235 is present in the amount of 0.71%, and has a half-life of 7.04×10^8 years and a specific activity of 1.54×10^4 pCi/g. U-230 has a short half-life of 20.8 days. Uranium exists in nature in two oxidation states, U^{+4} and U^{+6} (Hem 1985). Uranium is used primarily in the commercial nuclear power industry and in weapon systems (EPA 1985; Stokinger 1981). Depleted uranium (natural uranium depleted of uranium-235) is used in armor-piercing ammunition for the military, in inertial guidance devices and gyro compasses, as a counterweight for missile reentry vehicles, as radiation shielding material, and for x-ray targets (EPA 1985; USDI 1980).

Data on the transport and partitioning of uranium in the atmosphere are limited. Uranium compounds present in the atmosphere as particulates may be speciated, but their transport from air to surface water, plants, and soil will occur by wet and dry deposition. Therefore, the important factor in determining atmospheric transport will be particle size, distribution, and particle density.

In most waters, sediments will be a sink for uranium and the concentration of uranium in sediments and suspended solids will be several orders of magnitude higher than its concentration in water (Brunskill and Wilkinson 1987; Swanson 1985). The four most important factors controlling the mobility of

uranium from sediment to the water phase in natural waters are: oxidation-reduction potential (Eh), pH, characteristics of complexing agents or ligands, and the nature of sorbing materials in water.

The mobility of uranium in soil and its transport to other media depends primarily on properties of soil such as pH, oxidation-reduction potential, concentration of complexing anions, and sorption properties (Allard et al. 1982). The sorption of uranium to most soils is such that uranium may not leach readily from soil surface to groundwater, particularly in soils containing clay and iron oxide (Sheppard et al. 1987). Any soil property that alters the sorption mechanism will also alter the mobility of uranium in soil. Tetravalent uranium is relatively insoluble in soil-water systems, existing primarily as the solid species UO_2 across the entire spectrum of pH values, and reducing Eh values. When oxidized to the hexavalent form, uranium is highly mobile under all pH conditions, except for a limited area around pH 5 to pH 6.5 where the uranium carbonate is stable, precipitating as UO_2CO_3. At values greater than 6, the uranyl carbonate complex $UO_2(CO_3)_{2\text{-}2}(aq)$ becomes stable thereby increasing the solubility of uranium. At highly alkaline values, the highly soluble complex $UO_2(CO_2)_{2\text{-}4}(aq)$ is stable. Because uranium occurs in soils and fertilizers, the element is present in food and human tissues.

Vanadium

The majority of vanadium is used as an alloying agent (Hillard 1987). Vanadium compounds also have an important role as industrial catalysts. Vanadium-containing catalysts are used in several oxidation reactions such as the manufacture of phthalic anhydride and sulfuric acid. There are also used as corrosion inhibitors in flue-gas scrubbers.

From man-made sources almost all the vanadium released to the atmosphere is in the form of simple or complex vanadium oxides (Byerrum et al. 1974). Vanadium transported within the atmosphere is eventually transferred to soil and water on the earth's surface by wet and dry deposition (Duce and Hoffman 1976).

The transport and partitioning of vanadium in water and soil is influenced by pH, redox potential, and the presence of particulate. It has a natural concentration in groundwater ranging from less than 1 to 10 ppb (Dragun 1988). In water, vanadium generally exists in solution as the vanadyl ion (V^{+4}) under reducing conditions and the vanadate ion (V^{+5}) under oxidizing conditions, or as an integral part of, or adsorbed onto, particulate matter (Wehrli and Stumm 1989). The partitioning of vanadium between water and sediment is strongly influenced by the presence of particulate in the water. Vanadium is transported in water in one of two ways: solution or suspension. It has been estimated that only 13% is transported in solution, while the remaining 87% is in suspension (WHO 1988). Vanadium has a typical native soil concentration range of 20 to 500 parts per billion (ppb).

The mobility of vanadium in soils is affected by the pH of the soil. Relative to other metals, vanadium is fairly mobile in neutral or alkaline soils, but its mobility decreases in acidic soils (Van Zinderen Bakker and Jaworski 1980). Similarly, under oxidizing, unsaturated conditions some mobility is observed, but under reducing, saturated conditions vanadium is immobile (Van Zinderen Bakker and Jaworski 1980).

Zinc

Zinc is used most commonly as a protective coating for other metals. In addition, it is used in alloys such as bronze and brass, for electrical apparatus in many common goods, and in organic chemical extractions and reductions. It is also commonly used in batteries.

Zinc is present in the atmosphere as particulates, which may also contain other materials (Pacyna et al. 1989; Saltzman et al. 1985). Zinc-bearing particles from the atmosphere are transported to soil and water by wet (rain and snow) and dry deposition (gravitational settling and deposition on water and soil surfaces).

Zinc can occur in both suspended and dissolved forms in surface water. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability. Suspended (undissolved) zinc may be dissolved following minor changes in water chemistry or may be sorbed to suspended matter. The transport of zinc in the aquatic environment is controlled by the speciation of the ion. In natural waters, complexing agents, such as humic acid, can bind zinc. The stability of the zinc complex depends on the pH of the water and the nature of the complex. The tendency of zinc to be sorbed is affected not only by the nature and the concentration of the sorbent, but by pH and salinity as well (Callahan et al. 1979).

The mobility of zinc in soil depends on the solubility of the speciated forms of the compound and on soil properties such as cation exchange capacity, pH, redox potential, and species present in soil; under anaerobic conditions, zinc sulfide is the controlling species (EPA 1980; Kalbasi et al. 1978). Acidic conditions affect the release of zinc from soil to cationic exchange sites. Liming soil to increase pH or the addition of high amounts of inorganic matter reduces the bioavailability of zinc in soil. Zinc in a soluble form, such as zinc sulfate, is fairly mobile in most soils. However, relatively little land-disposed zinc is in the soluble form, and mobility is therefore limited by a slow rate of dissolution. Consequently, movement towards groundwater is expected to be slow, unless zinc is applied to soil in a soluble form or accompanied by corrosive substances (EPA 1980).

Zirconium

No information in the literature was found for the fate and transport of zirconium.

ORGANICS

Discussion of fate and transport properties for benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, phenanthrene, and pyrene are summarized as a group in the summary for polycyclic aromatic hydrocarbons. Most PAHs do not occur alone in the environment, rather they are found as mixtures of two or more PAHs. For this reason they will be discussed as a group.

1,1 Dichloroethylene

1,1-Dichloroethylene, also known as 1,1-dichloroethene and vinylidene chloride, does not occur as a natural product but is released into the environment as fugitive emissions and in wastewater during its production and use in the manufacture of plastic wrap, adhesives, synthetic fiber and as a chemical intermediate and solvent. It is used commonly as a coupling agent in antiknock gasoline, paint and varnish remover, metal degreaser, and ore floatation agent. Its largest industrial use is in the production of 1,1,1-trichloroethane.

In the atmosphere, the compound degrades by reaction with photochemically produced hydroxyl radicals, with a half-life of approximately 62 days for this degradation, and is typically scavenged by rainfall. Due to its moderate water solubility (5060 mg/L at 25 °C), the compound can be washed out of the atmosphere by rainfall.

If released on land, it will be removed rapidly by volatilization, although it may also leach into groundwater, where its fate is unknown, due to the compound's low adsorptivity. The organic content adsorption factor (K_{oc}) is 40 indicating little adsorption to soil organic matter. The compound is readily leached from landfill disposal sites. However, 1,1-Dichloroethylene has a high vapor pressure and low adsorption to soil and would therefore volatilize rapidly from soil.

If released in water, it will be removed by volatilization with a half-life of 6-9 days, 5-8 days, and 24-32 hr in a typical pond, lake, or river. Adsorption to sediment, biodegradation, and hydrolysis are relatively insignificant in the aquatic environment.

1,1-Dichloroethylene is known to undergo biodegradation in the presence of sewage. Bank filtration, the passage of river water through earth, is reported to reduce 1,1-dichloroethylene concentration by up to 25%. One study demonstrated biodegradation of 45-78% of the chemical within 7 days in the presence of wastewater; however, a significant portion of this loss was due to volatilization. Experiments to simulate the anaerobic conditions of groundwater biodegradation indicated this compound undergoes dechlorination to vinyl chloride. These experiments showed 50% of the compound disappeared in 5-6 months. Under simulated landfill conditions, degradation occurred in 1-3 weeks. The abiotic hydrolysis degradation half-life has been observed to be 6-9 months with no significant difference in hydrolysis rate between pH 4.5 and 8.5.

Bioconcentration in aquatic organisms will not be important.

1,1,1-Trichloroethane

1,1,1-Trichloroethane, also known as methyl chloroform, is not known to occur as a natural product and is likely to enter the biosphere from wastewater and stack emissions and fugitive emissions during its production. Also, volatilization is typical during cold cleaning of metals and other applications. The substance is also present in cleaning agents, pesticides, painted sheetrock, glued wallpaper and glued carpet.

1,1,1-Trichloroethane is fairly stable in the atmosphere and can be transported long distances in the atmosphere. It is slowly degraded principally by reaction with hydroxyl radicals and has a half-life of 6 months to 25 years. Due to the large input of 1,1,1-trichloroethane into the atmosphere and its slow degradation, the amount of 1,1,1-trichloroethane in the atmosphere is increasing by 12-17% per year. Some of this substance returns to earth in rainfall with tests indicating up to 40% atmospheric reduction as a result.

Due to its high vapor pressure (123.8 mm Hg at 25 °C), 1,1,1-trichloroethane evaporates fairly rapidly into the atmosphere. Yet it also passes rapidly through soil into groundwater. 1,1,1-trichloroethane has a high Henry's Law constant (8E-3 atm-m3/mole) and will volatilize rapidly from water and soil with diffusion through the liquid phase controlling volatilization from water. Half-life for volatilization from water obtained from laboratory systems range from a fraction of an hour to several hours. For water releases, it has been estimated the volatilization half-lives range from 5-10 days, 3-29 hours, and 4-12 days for ponds, rivers, and lakes, respectively.

The adsorption of 1,1,1-trichloroethane to soil is proportional to the organic carbon content of the soil. Experiments indicate that soil used to bank-filter 1,1,1-trichloroethane-contaminated water does not retain much of the contaminant. In addition, 1,1,1-trichloroethane is frequently found in groundwater in high concentrations; therefore, it can be concluded that 1,1,1-trichloroethane is not strongly adsorbed by soils, especially subsurface soils.

1,1,2-Trichloro-1,2,2-Trifluoroethane

No information in the literature was found for the fate and transport of 1,1,2-Trichloro-1,2,2-Trifluoroethane.

1,1,2,2-Tetrachloroethane

1,1,2,2-tetrachloroethane in the environment is expected to be moderately volatile from aqueous solutions, weakly sorbed to soils, and has a low potential for bioaccumulation in living organisms. Although it is likely to volatilize from soil surfaces, the fraction not subject to volatilization is expected to be fairly mobile in groundwater (ADL 1986).

1,1,2,2-Tetrachloroethane has a K_{oc} of 46 in a silt loam soil and the partitioning to a low organic carbon soil was 0.05 which suggests that it will not absorb appreciably to soil, suspended solids, and sediment. Sorption, however may be significant in dry soils with high clay content. It would be expected to readily leach from soil surfaces into the subsurface soil and groundwater (ATSDR 1989). 1,1,2,2-Tetrachloroethane is expected to have limited persistence and mobility in surface soils. In surface soils, 1,1,2,2-tetrachloroethane is expected to sorb to soil, with only a small fraction partitioning into the soil-water or soil-air phase. In deeper soil, however, 1,1,2,2-tetrachloroethane is expected to partition equally between soil and water. Transport of 1,1,2,2-tetrachloroethane vapors through air-filled pores in soil is expected to be an important removal mechanism for this contaminant. Its K_{ow} value (245) indicates weak absorption to soils or organic matter.

1,2,4-Trichlorobenzene

No information in the literature was found for the fate and transport of 1,2,4-Trichlorobenzene.

1,3-Dinitrobenzene

The important fate processes for 1,3-DNB are uncertain. Simmons and Zepp (1986) determined that the average annual rate constant for the direct photolysis of 1,3-DNB in distilled water was 0.03 $(day)^{-1}$ at 40°N latitude. Spanggord et al. (1980a) noted a 50% loss of the 1,3-DNB component of condensate water after 12 days of exposure to sunlight. Therefore photolysis may represent a significant fate process. Volatilization is probably not an important loss mechanism for 1,3-DNB. Physical transport of 1,3-DNB from aqueous systems is believed to be unimportant because of its low vapor pressure of $1.31*10^{-4}$ at 25°C (Maksimov 1963). In addition, its Henry's law constant of $5.44*10^{-8}$ at 25°C (Burrows et al 1989) indicates little tendency for volatilization from water. Soil adsorption data was not found, however, due to the low sediment adsorption coefficient of 36.31 (Lyman and Loreti 1987); 1,3-DNB is not expected to remain in soils for extended periods of time.

Bioaccumulation is probably not an important transport mechanism for 1,3-DNB. Isnard and Lambert (1988) determined the BCF for 1,3-DNB in fish to be 4.7. No other data pertaining to bioaccumulation for 1,3-DNB were found during completion of this profile.

1,3,5-Trinitrobenzene

The major environmental fate processes for 1,3,5-TNB are not well documented. Studies by Burlinson (1980, 1973a, 1973b) suggest that photolysis may not be a significant transformation process. Physical transport of 1,3,5-TNB from aqueous systems is believed to be unimportant because of its low vapor pressure, $3.03*10^{-6}$ at 25°C (Burrows et al. 1989). In addition, its Henry's law constant of

$2.21*10^{-9}$ at 25°C indicates little tendency for volatilization from water. Sorption of 1,3,5-TNB from aqueous systems is believed to be unimportant because of its low sediment adsorption coefficient of 19.95 (Burrows et al. 1989).

Isnard and Lambert (1988) report a BCF of 2.65 in fish, which indicates only a very slight tendency for 1,3,5-TNB to accumulate in aquatic life. Trabalka and Garten (1982) reported that chemicals with log K_{ow} values less than 3.5 or solubilities greater than 10 mg/L do not accumulate in mammals or birds. Therefore, 1,3,5-TNB which has a K_{ow} of 15.14 and a solubility value of 385 mg/L, would not tend to accumulate in animal receptors in significantly high concentrations.

1,4-Dichlorobenzene

One major consumptive use of p-DCB is as a space deodorizer for toilet bowls, urinals, garbage cans, and diaper pails. These products are usually 100% p-DCB blocks, sometimes with added perfume. A second important use of p-DCB is as a chemical intermediate in the production of PPS resins (ICF 1987).

Theoretically atmospheric p-DCB may be degraded by chemical or sunlight catalyzed reactions or may be absorbed onto particles that settle or are removed from the atmosphere by rain. Dichlorobenzenes in general were reported by Ware and Weast (1977) to be reactive with hydroxyl radicals in the air with a half-life of approximately 3 days.

The available data indicate that degradation of chlorobenzenes in aquatic systems may be carried out by microbial communities in wastewater treatment plants and in natural bodies of water (EPA 1985). The available data also indicate that p-DCB probably volatilizes from the water column to the atmosphere at a relatively rapid rate.

1,4-Dichlorobenzene can be quite persistent in soils, studies indicate that these compounds are somewhat resistant to microbial degradation. The only case where 1,4-dichlorobenzene was easily degraded was where the soil was already enriched (Monsanto 1986).

p-DCB has a high potential for bioaccumulation in aquatic organisms. The incorporation of chlorine into an organic molecule generally increases its lipophilic character and bioaccumulation potential (Kopperman et al. 1976).

2-Butanone

2-Butanone is produced on a commercial scale by one of two processes. The vapor-phase dehydrogenation of sec-butanol (2-butanol), itself obtained from the hydrolysis of butene, accounts for 88% of 2-butanone production (Neier and Strehlke 1985; Papa and Sherman 1981). In the other commercially significant process, 2-butanone is obtained as a byproduct of acetic acid production. In this methodology, liquified butane is subject to catalytic oxidation. 2-Butanone exhibits outstanding solvent properties, and combined with its low cost, it is often the choice solvent for various coating systems (Neier and Strehlke 1985; Papa and Sherman 1981). It is used in the degreasing of metals, as an extraction solvent, in dewaxing applications, and as a solvent for the production of smokeless powders.

In the atmosphere, 2-Butanone is expected to exists predominantly in the vapor phase (Eisenreich et al. 1981; Riddick et al. 1986). The short residence time expected for 2-butanone in the atmosphere, less than 1 day, suggests that it is not transported long distances from the original point of release.

2-Butanone (methyl ethyl ketone) is fairly mobile in soil/groundwater systems. 2-Butanone is highly soluble in water as evidenced by its very low K_{oc} and K_{ow} values. 2-Butanone is slightly less mobile in soil/groundwater systems than acetone. Since acetone is not strongly sorbed to soils, some volatilization may also occur. Acetone is fairly susceptible to microbial degradation (ADL 1987). If 2-butanone is released to water it is expected to rapidly volatilize to the atmosphere based on its K_{ow} of 3.55.

2-Chlorotoluene

Limited fate and transport information was found for 2-Chlorotoluene more commonly called o-chlorotoluene. It is volatile with steam. Slightly soluble in water but freely soluble in alcohol, benzene, chloroform, and ether. It is flammable when exposed to heat or flame. When heated to decomposition, it emits toxic fumes of Cl ions.

Information was found for a similar compound, 4-chloro-1-methylbenzene (CAS 106-43-4). 4-Chloro-1-methylbenzene, also called 4-chorotoluene or p-chlorotoluene, has the same molecular formula and weight as o-chlorotoluene; however, though the methyl group is located in the same position (position 1) of the benzene ring, in p-chlorotoluene the Cl ion is attached in position 4 (as opposed to position 2 for o-chlorotoluene). The following is a summary of p-chlorotoluene fate and transport information.

Based on the vapor pressure, p-chlorotoluene is expected to exist almost entirely in the vapor phase in the ambient atmosphere. The dominant removal mechanism is expected to be reaction with photochemically generated hydroxyl radicals which has an estimated half-life of 8.4 days. A slight potential exists for direct proteolysis in the atmosphere. Physical removal from air via washout may occur; however, volatilization will probably return the p-chlorotoluene to the atmosphere.

If released to soil, p-chlorotoluene is expected to leach slowly with a relatively low soil mobility. Volatilization may occur fairly rapidly from dry soil surfaces due to its relatively high vapor pressure (2.59 mm Hg at 20 °C). Chemical hydrolysis should not be environmentally important. Soil adsorption coefficients have been estimated at K_{oc} of 446-1544, indicating p-chlorotoluene would have low mobility in soil and that adsorption to suspended solids and sediments in water may have some environmental significance.

The value of the Henry's Law constant for p-chlorotoluene (0.00407 atm-m3/mol, calculated from the vapor pressure and water solubility) suggests that volatilization is probably significant from all bodies of water. If released to water, volatilization (half-life of 3.5 hours based on modeling results), sensitized photolysis, and adsorption to suspended solids and sediments are potentially important fate processes. The relative importance of these fate processes and the rate of compound loss are expected to vary depending on ambient conditions and characteristics of the water body. This compound is not expected to undergo chemical hydrolysis, react with oxidants found in natural waters or bioaccumulate significantly in aquatic organisms.

Due to a lack of data, the significance of biodegradation in soil is not known. One screening test indicated that p-chlorotoluene is resistant to biodegradation as determined by the theoretical biological oxygen demand. Based on monitoring data from the River Rhine, the half-life of p-chlorotoluene in a river 4-5 m deep during midsummer was estimated to be 1.2 days. p-Chlorotoluene is inert to chemical hydrolysis under environmental conditions. Reaction of p-chlorotoluene with free radical oxidants found in natural waters is not an environmentally important fate process.

Based on the water solubility and other factors, the bioconcentration factor for p-chlorotoluene has been estimated to range from 45-200, suggesting that slight bioaccumulation in aquatic organisms may occur.

2-Hexanone

2-Hexanone is not currently manufactured, processed, or used for commercial purpose in the United States (EPA 1987). 2-Hexanone has been used as a solvent for many materials, primarily in the lacquer industry as a solvent for lacquers and varnish removers. It has also been used as a solvent for ink thinners, resins, oils, fats, and waxes. 2-Hexanonehas also been used as an intermediate in the synthesis of organic chemicals (ACGIH 1986; HSDB 1989).

2-Hexanone exists in the atmosphere as a vapor. Liquid 2-Hexanone is volatile; its vapor pressure has been measured as $1.53 * 10^{-2}$ atm (11.6 mm Hg) at 25 °C (Ambrose et al. 1975). Because 2-hexanone is very soluble in water, a large fraction of 2-hexanone released to the atmosphere may dissolve in water vapor (such as clouds and rain drops).

2-Hexanoe is very soluble in water. 2-Hexanone will volatilize from water more rapidly from rivers than from lakes or ponds. There is no information on whether 2-hexanone in water is expected to partition to soils and sediment.

2-Hexanone will probably not be concentrated by organisms in water and thus will not be an important fate mechanism.

2-Methylnaphthalene

No information in the literature was found for the fate and transport of 2-Methylnaphthalene.

2-Nitrophenol

No information in the literature was found for the fate and transport of 2-Nitrophenol.

2-Propanol

2-Propanol is also called isopropanol or isopropyl alcohol. It is present naturally in plant volatiles, microbial degradation of animal waste, and emissions from volcanoes. Emissions from anthropogenic sources include petroleum storage, auto exhaust, plastics combustion, printing, sewage treatment, wood pulping, and steel manufacturing. It is also a common consumer solvent.

In the atmosphere it will photodegrade primarily by reaction with hydroxyl radicals with a half-life of one to several days. The estimated half-life of isopropanol in the atmosphere ranges from 3.5 days to 33 hr. Alcohols are known to be resistant to hydrolysis.

Isopropanol will enter the environment as emissions from its manufacture and use as a solvent. When released on land it is apt to both volatilize and leach into groundwater quickly due to its high vapor pressure and low adsorption to soil. In the soil and groundwater it may possibly biodegrade but no data on the rates of these processes are known. If soil degradation is not rapid, it is apt to leach into the groundwater. Its fate in groundwater is unknown.

When released in water, isopropanol will volatilize and biodegrade. The volatilization half-life from a body of water is estimated to be approximately 5.4 days. It would not be expected to adsorb to sediment or bioconcentrate in fish. Although no data on its rate of degradation in natural waters could be found, laboratory tests suggests that it is not very long-lived in water. Isopropanol was 99% degraded with acclimated activated sludge at 20 °C.

No information on the bioconcentration factor for isopropanol could be found in the literature. Its low octanol/water partition coefficient indicates that it will not bioconcentrate in aquatic organisms.

2,3,7,8-Tetrachloro dibenzodioxin

Chlorinated dibenzo-p-dioxins are derived from dibenzo-p-dioxin with from 1-8 Cl atom substitutions for H atoms, giving a total of 75 possible chlorinated derivatives commonly referred to as dioxins and of which 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is the most notable. TCDD, which is one of the most toxic of all synthetic substances to some animals, was produced as a low-level contaminant in the manufacture of some aromatic, oxygen-containing organohalide compounds such as chlorophenoxy herbicides and hexachlorophene manufactured by processes used until the 1960s. It is also formed during various combustion processes. Incineration of chemical wastes, including chlorophenols, chlorinated benzenes, and biphenyl ethers, may result in the presence of TCDD in flue gases, fly ash, and soot particles. The chlorophenoxy herbicides, such as 2,4,5-trichlorophenoxy-acetic acid (2,4,5-T), were manufactured on a large scale for weed and brush control and as military defoliants.

TCDD has a very low vapor pressure of only 1.7E-6 mm Hg at 25 °C and a water solubility of only 0.2 µg/L. Low water soluble chemicals partition to soil or sediments and bioconcentrate in aquatic organisms, may volatilize more readily from water (depending on the chemical's vapor pressure), and are less likely to be biodegradable. The biodegradation of TCDD in water is probably slow. The two processes that may be important for the removal of TCDD are volatility and photodegradation. The volatility of a chemical is dependent upon the vapor pressure; low vapor pressures result in the chemical being associated mostly with particulate matter. It has a high degree of chemical stability, and is poorly biodegradable.

TCDD is expected to be immobile in most soils by irrigation and rainfalls. The estimated half-life of TCDD on soil surfaces is one to three years, but the half-life in the interior of soil may be 10 to 12 years (EPA 1985). TCDD has the potential for bio-accumulation in animals.

2,4-Dimethylphenol

2,4-Dimethyphenol, also called 2,4-xylenol, is released to the environment as fugitive emissions and in wastewater as a result of coal tar refining, coal processing, and in its use in chemical/plastics manufacturing. It may be released during its use in manufacture of plastics and resins, pharmaceuticals, insecticides, fungicides, disinfectants, and solvents. It may be released in asphalt and roadway runoff, domestic sewage, gasoline and diesel exhaust, and tobacco smoke. Dimethylphenols have also been identified as naturally-occurring constituents of some plants.

In the atmosphere, vapor phase 2,4-dimethyphenol will degrade during daylight hours by reaction with photochemically produced hydroxyl radicals (half-life 8 hr). At night it will probably degrade very rapidly by reaction with nitrate radicals. Washout by rain will also be an effective removal process.

When released in water, it will degrade principally due to biodegradation with a half-life of hours to days. In humic waters, oxidation by alkyl peroxy radicals may also be important. Adsorption to

sediment and particulate matter in the water column will only be moderate and bioconcentration in fish should not be significant. The half-life should be less than several days in humic waters due to photooxidation by alkylperoxy radicals. Photolysis may occur in clear surface waters. Because of the low Henry's Law Constant (6.4E-7 atm-m^3/mole at 8 °C), volatilization from water would not be a significant transport process.

If spilled on soil, 2,4-dimethyphenol will probably adsorb moderately to the soil and biodegrade in several days. No experimental data on the adsorption of 2,4-dimethyphenol to soil is available.

96% removal in 5 days was achieved in screening studies using activated sludge and 100% degradation occurred in 7 days with a sewage starter.

2,4-Dichlorophenoxyacetic acid

2,4-Dichlorophenoxyacetic acid, also known as 2,4-D, is commonly released to the environment through its use in systemic herbicide formulations and as a hydrolysis product of 2,4-D esters or from spills. There is no known natural source. Environmental pollution with 2,4-D may also occur as a result of the production and disposal of 2,4-D or of its byproducts and discharge of treated or untreated industrial effluent. Disposal of unused 2,4-D and washing of equipment may result in localized land or water pollution.

The primary source of 2,4-D in the atmosphere is spray applications of the herbicide or its mixtures. Spray drift is capable of carrying it up to a few kilometers. If released in air it will be subject to photooxidation (estimated half-life of 1 day). Gravitational settling of aerosol and rainout (due to its significant solubility in water) may also be significant removal processes.

If released on land it will probably readily biodegrade (typical half-lives <1 day to several weeks). Biodegradation is by far the most important loss process for 2,4-D in most soils, leading to various hydroxylic aromatic products. The rate of degradation is affected by several conditions, especially the concentrations of 2,4-D and water content, temperature and the organic content of soil and the status of preexposure of the soil to 2,4-D or its esters. Typical half-lives range from less than a day to several weeks. Dryer, sandy soils with low organic content lead to longer biodegradation half-lives. Its adsorption to soils will depend on organic content and pH of soils (pKa of 2,4-D = 2.64-3.31), but it will not be expected to appreciably adsorb to soils. Leaching groundwater will likely be a significant process in coarse-grained sandy soils with low organic content or with very basic soils. Adsorption appears to increase with increasing organic content and decreasing pH of soil. Evaporation and hydrolysis will be negligible.

If released to water it will be lost primarily due to biodegradation (typical half-lives 10 to >50 days). It will be more persistent in oligotrophic waters and in waters where high concentrations are released. The degradation rate is especially dependent upon level of nutrients present, temperature, availability of oxygen, and whether or not the water has a prior history of contamination by 2,4-D or other phenoxyacetic acids. Degradation will be rapid in sediments (half-life <1 day). It will not bioconcentrate in aquatic organisms or appreciably adsorb to sediments, especially at basic pHs. Volatilization from water and soil is expected to be negligible based on its extremely low reported Henry's Law constant (1.02E-8 atm-cm^3/mol) and hydrolysis will be negligible.

There are a variety of microorganisms in soil, freshwater and marine ecosystems which are capable of degrading 2,4-D. The rate will depend on a number of factors including presence of acclimated

organisms, nutrient levels, temperature and concentration of 2,4-D. Half-lives in rivers of 18-50 days in clear water and 10-25 days in muddy water have been measured.

Estimation of the bioconcentration factor (BCF = 7) indicates there is little tendency for 2,4-D to bioconcentrate in aquatic organisms.

2,4-Dinitrotoluene

2,4-Dinitrotoluene (2,4-DNT), also known as 1-methyl-2,4-dinitrotoluene, is used to prepare 2-4-diaminotoluene, an organic synthesis, and to make dyes, explosive, and propellant additives. 2,4-DNT (2,4-dinitrotoluene) may enter the environment in wastewater from the processes in which it is made and used.

In the atmosphere, 2,4-DNT is estimated to have of half-life of 8 hours, degrading by vapor phase reaction with photochemically produced hydroxyl radicals.

In soil, 2,4-DNT will be slightly mobile. Aromatic nitro compounds are not susceptible to hydrolysis and photolysis should not be an important process in soil. Some biodegradation may occur in both aerobic and anaerobic zones of soil. The estimated K_{oc} value and the measured sediment adsorption coefficient indicate that 2,4-DNT will have a slight tendency to sorb to sediments, suspended solids, and biota.

The volatilization half-life from bodies of water is estimated to be between 1-8 years, indicating volatilization from water is not a significant transport mechanism. Photolysis is probably the most significant removal process for 2,4-DNT in water, with half-lives for this mechanism estimated to be 2.7, 9.6, and 3.7 hours for rivers, bays, and ponds, respectively.

Tests indicate 2,4-DNT did not degrade in an aerobic batch culture after 14 days of incubation. Municipal sludge organisms were inhibited by 2,4-DNT concentrations as low as 10 mg/L. Anaerobic conditions appear to benefit biodegradation. Bioconcentration factors (BCFs) range from 13-2000 for various microorganisms and algae. Fish studies indicate a BCF range of 4-103, indicating 2,4-DNT does not bioconcentrate much in aquatic organisms.

2,4,6-Trinitrotoluene

In aqueous environments, 2,4,6-TNT is degraded primarily through photolysis and secondarily through hydrolysis (Spanggord et al. 1981). Half-lives in soil have been reported as ranging from 4 to 10 years, with the microbial oxidative decomposition rate varying from 0.05 to 0.017 percent per day (Cataldo et al. 1989).

The extent of soil sorption is significant, and positively correlated with both pH and temperature (Kayser and Burlison 1988; Sikka et al. 1980). Cataldo et al. (1989) observed that the 2,4,6-TNT uptake rate in bush bean, wheat, and blando brome was approximately 60 micrograms/hour/gram fresh weight root. These studies suggest that 2,4,6-TNT becomes incorporated into plants as polar metabolites. Toxic concentrations range from 1 ppm duckweed (Schott and Worthley 1974) to 30 ppm in bean, wheat, and blando brome (Cataldo et al. 1989). A bioconcentration factor of 19 suggests that bioaccumualation is not a significant fate process (Dames & Moore1991).

2,6-Dinitrotoluene

Photolysis, sorption, and biodegradation are the most probable fate processes for 2,6-DNT. Photolysis in solution may be a highly probable fate process for 2,6-DNT (Callahan et al. 1979). The photolysis half-life of 2,6-DNT in pure water is 42 hours (Spanggord et al. 1980). Simmons and Zepp (1986) found that dissolved or suspended humic substances greatly enhance (10 to 17 times) indirect photolysis of 2,6-DNT (USPHS 1989c). The ability of polynitroaromatic compounds to form highly stable charge-transfer complexes with more highly electronegative aromatic compunds (Hall and Poranski 1970) indicates that 2,6-DNT should be strongly adsorbed by both humus and clay. Also, its log octanol-water partition coefficient (K_{ow}) of 2.05, calculated by the method of Tute (1971), is sufficiently large to indicate significant adsorption of 2,6-DNT by humus (Callahan et al. 1979). However, leaching may occur and, therefore, adsorption to soil and sediment may be very minor (Burrows et al. 1989). In the absence of sunlight and oxygen, any losses of DNTs would apparently be dependent on biodegradation (USPHS 1989c). It has been reported that dinitrotoluenes decompose very slowly in a reservoir (Galuzova 1963). More recently, Spanggord et al. (1980) observed biodegradation of DNTs in an aerobic environment, with a half-life of less than one hour. Bioaccumulation may not be an important process for 2,6-DNT. This contention is supported by 2,6-DNT's relatively low log K_{ow} of 2.05(Callahan et al. 1979).

4-Chloroaniline

Also called p-chloroaniline and 1-amino-4-chlorobenzene, 4-chloroanaline is released to the environment as fugitive emissions, in wastewater during its production, or use as a dye intermediate or in the manufacture of pharmaceuticals and agricultural chemicals.

Degradation in air will primarily be due to reaction with hydroxyl radicals (half-life of 4.6 hr) although direct photolysis is also possible.

If released on soil, it will rapidly combine chemically with soil components and partially be mineralized by chemical and biological action. A few percent of the 4-chloroaniline will volatilize from the soil.

If released to water, 4-chloroanaline will be primarily lost due to volatilization (half-life about 6.4 hours), photooxidation in surface layers (half-life of 0.4 hour), and rapid chemical reactions with humic materials and clay in the water column and sediment.

4-Methylphenol

4-Methylphenol, is commonly referred to as 4-cresol but is also called p-methylphenol, p-cresylic acid, and 4-hydroxytoluene, among other names. Cresols like this one occur naturally in petroleum and plant volatiles. 4-Methylphenol is produced anthropogenically from coal tar refining and is used as a disinfectant and for metal refining and chemical manufacturing. It is also found in automobile engine emissions, wood pulp, glass fiber manufacturing, and tobacco smoke. Additionally, 4-methylphenol is a byproduct of toluene photooxidation.

The photochemical half-life of 4-methylphenol during daytime is approximately 10 hr while at night it is more on the order of 4 minutes. The dominant reactions are with hydroxyl radicals during daytime and nitrate radicals at night. Smog conditions reduce the daytime half-life. 4-Methylphenol is highly soluble in water and will likely be scavenged from the atmosphere by rainfall.

4-Methylphenol is relatively mobile in some soils and as a result is likely to leach to groundwater. It biodegrades rapidly in soil with tests indicating complete degradation in about one week. The K_{oc} value has been measured experimentally in clay loam as 49. Free iron oxide and pH appear to control soil adsorption of 4-methylphenol.

Biodegradation appears to be the dominant loss mechanism for aquatic releases of 4-methylphenol. Modeling indicates degradation half-lives range from 0.55 hr to 2400 hr for rivers and lakes, respectively. The lake degradation appears to decrease with degree of eutrophication. Marine water releases result in degradation half-life range of 9-43 hr. Degradation half-life under anaerobic conditions appears to be on the order of weeks. 4-methylphenol has low volatility from water (Henry's Law constant = 9.6E-7 atm-m^3/mol); the evaporation half-life from water has been estimated as 1.4 yr. and 290 yr. for rivers and lakes, respectively. This is much slower than the biodegradation rates. Groundwater downgradient of a wood preservative facility was found to be disproportionately low in 4-methylphenol concentration based on the substance's release rate. The disproportion is attributed to rapid biodegradation.

The bioconcentration factor for 4-methylphenol has been estimated as 18; this indicates a low probability of bioconcentration in organisms.

4-Chloro-3-methylphenol

4-Chloro-3-methylphenol, also called p-chloro-m-cresol, is a white or slightly pink crystalline substance with a phenolic odor that is readily volatile in steam and organic solvents and somewhat soluble in water (1:250) at 25 °C. It has a melting point of 66 °C and a boiling point 235 °C. It is incompatible with sodium hydroxide and when heated to decomposition it emits toxic fumes of Cl^- and phosgene.

It is an irritant to skin. It is used as an external germicide and a preservative for glues, gums, paints, inks, textile, and leather goods. No further fate and transport information is available.

Acenaphthene

See discussion for PAH.

Acetone

Acetone is used primarily as an intermediate in chemical production and as a solvent (Nelson and Webb 1978). In 1989, 40% of the available acetone was used in the production of methyl methacrylate, methacrylic acid, and higher methacrylates; 20% was used as solvent; 13% was used in the production of bisphenol A; 10% was used in the production of methyl isobutyl ketone and methyl isobutyl carbinol; 6% was used in drug and pharmaceutical applications; 5% accounted for miscellaneous uses; and 6% was exported (CMR 1990).

Since the vapor pressure of acetone is 181.72 mm Hg at 20°C, acetone should exist exclusively in the vapor phase in the atmosphere. Due to the atmospheric half-life, which is on the order of days, acetone will be transported long distances in the air. Because it is completely miscible in water, acetone will be removed from the atmosphere by wet deposition (Grosjean and Wright 1983), which will transport acetone from the atmosphere to surface water and soil.

Acetone is expected to be mobile in soil/groundwater systems. Only a small amount of acetone released into the environment is expected to sorb to soil. The majority is expected to migrate freely through the soil-water phase and migrate freely via diffusion and infiltration with little or no retardation.

Acetone is infinitely soluble in water as evidenced by its very low K_{oc} and K_{ow} values. Since acetone is not strongly sorbed to soils, some volatilization may also occur. Leaching transports acetone from soil to groundwater. The rate of leaching from soil by rainwater depends on the sorption characteristics of acetone soil. Since acetone has a low K_{oc} value, sorption of acetone in soil will be weak. The low retention ability will permit acetone to leach to the groundwater. Acetone is highly susceptible to microbial degradation (ADL 1987).

Acetonitrile

Acetonitrile, also called methyl cyanide, is typically released to the environment during its manufacture and use, from shale oil retorting and coal gasification, incineration of polyacrylonitrile, from automobile exhaust, cigarette smoke, and turbine engines. Acetonitrile has been detected in shale oil wastewaters and in wastewater from coal gasification process.

Acetonitrile is likely to be unreactive towards direct photolysis in air and the half-lives for its reaction with OH^- radicals and ozone have been estimated to be 535 days and 860 days, respectively. Therefore, it will persist in the troposphere for a long time and may be transported long distances from its source of emission. Wet deposition may remove some of the atmospheric acetonitrile.

If released to soil, aerobic biodegradation is likely to occur in subsoil. Acetonitrile is expected to be mobile in soil and may evaporate from soil surfaces. It has high water solubility, moderately high vapor pressure, and weal soil sorption suggesting volatilization from soil surfaces and leaching into groundwater may readily occur.

Biodegradation is expected to be a major loss process in water. Acclimatization increases the biodegradation rate substantially as long as the acetonitrile concentration is not too high (i.e., 500 mg/L). The decomposition of the compound in the Ohio River water was measured as 20% in 5 days and 40% in 12 days.

Volatilization may become competitive with other loss processes, particularly at shallow water depths. Hydrolysis, photolysis, adsorption to suspended particles and sediments and bioconcentration in aquatic organisms are not likely to be important. The volatilization half-life of acetonitrile is estimated as 21 hours for river water, based on the compound's Henry's Law constant and an arbitrary wind speed of 3 m/s.

Acrylonitrile

Acrylonitrile, also known as 2-propenenitrile, is not a naturally occurring substance but is a very high production chemical which may be released to the environment as fugitive emissions or in wastewater during its production and use in the manufacture of acrylic and modacrylic fibers, ABS resins, adiponitrile, acrylamide, and other chemicals and resins. It is also found in auto exhaust and clothing and furniture. Acrylonitrile is also released during the burning of polyacrylonitrile plastic and tobacco products.

If released to the atmosphere, acrylonitrile will degrade by reaction with hydroxyl radicals with a reaction half-life of about 40 hours of sunlight. This is considerably reduced in the presence of smog. The relatively long atmospheric half-life indicates considerable dispersion would be likely.

If spilled on land, acrylonitrile will evaporate rapidly. It is known to not adsorb well to soil (K_{oc} of 9); therefore, may leach to groundwater. If released into water, acrylonitrile biodegrades slowly. Half-

lives for biodegradation in aquatic settings range from 1-3 weeks depending on the degree of microbial acclimatization. Volatilization from water is expected to occur at half-lives of 1-6 days. Acrylonitrile will undergo photooxidation in humic water may also occur.

The whole body bioconcentration factor for fish has been experimentally determined as 48 indicating that the bioconcentration of acrylonitrile in aquatic organisms is not significant.

Anthracene

See discussion for PAH.

Benzene

Benzene is considered to be highly volatile with a vapor pressure of 95 mm Hg at 250 °C. Benzene is soluble in water with a solubility of 1,780 mg/L at 250 C. Benzene released to soil surfaces partitions to the atmosphere through volatilization, to surface water through runoff, and to groundwater as a result of leaching.

Benzene has a K_{oc} of 60-83 and is thus considered highly mobile. Benzene is expected to be fairly mobile in soil-groundwater systems. Transport with infiltrating water is likely, particularly in sandy soils and soils with low organic matter content. Volatilization of benzene in near-surface soils is expected to be high. Results show that benzene partitions mainly into the air (99%), with less than 1% partitioning into water, soil, sediment suspended sediment, and biota. Transformation processes, including hydrolysis and biodegradation, are not expected to substantially decrease environmental levels of benzene. Its K_{ow} value (135) indicates weak adsorption to soils or organic matter. These data indicate that benzene is highly volatile, weakly absorbed to soil, mobile in soil-water systems, and not likely to bioaccumulate in living organisms (ADL 1986).

Since benzene is not very lipophilic, reported log K_{ow} of 1.95 and an estimated BCF of 24, benzene is not expected to bioconcentrate to any great extent in aquatic organisms (ATSDR 1992b). Under aerobic conditions, benzene is biodegraded in soil. Microbial metabolism of benzene proceeds through the formation of cis-dihydrodiols and, with further metabolism, to catechols which are the substrates for ring fission.

Benzine

No information in the literature was found for the fate and transport of benzine.

Benzo(a)anthracene

See discussion for PAH.

Benzo(a)pyrene

See discussion for PAH.

Benzo(b)fluoranthene

See discussion for PAH.

Benzo(k)fluoranthene

See discussion for PAH.

Benzo(g,h,i)perylene

See discussion for PAH.

Butyl alcohol

No information in the literature was found for the fate and transport of butyl alcohol.

Butylbenzylphthalate

Butylbenzylphthalate is used as a plasticizer for polyvinyl and cellulosic resins, primarily in PVC. It is commonly used to make flooring materials.

BBP released to the atmosphere has an estimated half-life of 1-5 days. Volatilization of BBP to the atmosphere is not expected to be a significant transport mechanism since BBP has a low vapor pressure of 8.6E-6 mm Hg at 20 °C.

If released on land, butylbenzylphthalate (BBP) adsorbs fairly well to soil (measured adsorption coefficient ranges from 68-250) and will not be expected to leach to groundwater significantly although it has been detected in groundwater. The most significant fate process for BBP in soil is degradation. BBP has low volatility and does not rapidly evaporate from soil.

BBP released to water will partition to solids such as sediment and biota. The primary fate mechanism for BBP in water is biodegradation. Primary degradation of >95% BBP in water occurred in about 7 days with near total degradation occurring in about 28 days. Based on its estimated Henry's Law constant (<1E-6 atm-m^3/mol), volatilization from water will not be significant except for shallow rivers or during high wind activity. Photodegradation and hydrolysis in water (half-lives of >100 days for BBP) are not significant loss mechanisms for BBP in water.

Biodegradation appears to be the primary loss fate mechanism for BBP. BBP is readily biodegraded in activated sludge, semicontinuous activated sludge, salt water, lake water, and under anaerobic conditions. Biodegradation by mixed microbial cultures is fairly rapid with one test indicating 99% BBP reduction from an initial concentration of 3.3 mg/L within 48 hours. Anaerobic degradation occurs on the order of weeks.

Carbon disulfide

Carbon disulfide, also known as dithiocarbonic anhydride, appears naturally as a result of biological activity in oceans; emissions from oceans total have been estimated as 6E+11 g/yr., with coastal areas and other areas of high biological productivity having greater fluxes of carbon disulfide than open oceans. Other naturals sources include sulfate reduction in soils, volcanic emissions, and marshlands. Anthropogenic sources of carbon disulfide releases include emissions and wastewater during carbon disulfide production for the manufacture of viscose rayon, carbon tetrachloride, cellophane, and other regenerated celluloses and rubber chemicals; carbon disulfide is also used as a solvent. Furthermore, sewage treatment plants, landfills, municipal sludges, and coal tar are known to contain some carbon disulfide. It is also used for insect control in stored grains as well as an herbicide and

fungicide. Total global input of anthropogenic carbon disulfide is estimated to be 36E+10 g/yr. and arises principally from the chemical industry and sulfur recovery processes.

If released into the atmosphere, carbon disulfide reacts with atomic oxygen and photochemically produced hydroxyl radicals with a half-life of around 9 days. The action of soil in adsorbing and degrading gaseous carbon disulfide demonstrates that soil may be natural sink for carbon disulfide.

If released on land, carbon disulfide will be primarily lost due to volatilization. The K_{oc} for carbon disulfide has been estimated as 63; therefore, it is not expected to adsorb significantly to soil. Experiments indicate air-dried soil can adsorb up to 50% of carbon disulfide released; soils at 50% water-holding capacity showed much less adsorption initially. With time, the adsorption rates of carbon disulfide in air-dried soils appear to be less than wet soils; however, evidence indicates the moist soil adsorption only occurs in unsterilized soils suggesting that this adsorption is really the result of microbial activity. Since it has a low adsorptivity to soil, it should readily leach to groundwater; evidence exists to suggest that subsurface biodegradation of carbon disulfide will occur.

If released into water, carbon disulfide will be primarily lost due to volatilization (modeling based on an estimated Henry's Law constant of 0.0014 atm-m3/mol for carbon disulfide indicates a half-life of 2.6 hr for volatilization from rivers). Adsorption to sediment should not be significant.

Carbon disulfide has been classified as difficult to biodegrade. Tests indicate carbon disulfide can persist in unadapted soils for 3 months to a year. The bioconcentration factor for carbon disulfide has been estimated from carbon disulfide's water solubility as 7.9 indicating carbon disulfide would not be expected to bioconcentrate much in aquatic organisms. Abiotic photolysis degradation is not considered to be a significant loss mechanism for carbon disulfide. Based on source estimates and global burdening, it is estimated that the half-life of carbon disulfide in the troposphere is somewhere on the order of 9 days.

Carbon tetrachloride

Carbon tetrachloride is produced by exhaustive chlorination of a variety of low molecular weight hydrocarbons such as carbon disulfide, methanol, methane, propane, and ethylene dichloride (CEH 1985; IARC 1979). It is also produced by thermal chlorination in the production of tetrachloroethylene. The major current use of carbon tetrachloride is in the production of chlorofluorocarbons, such as dichlorodifluoromethane (F-12) and trichlorofluoromethane (F-11), that are used primarily as refrigerants (CEH 1985). Because carbon tetrachloride is a powerful solvent, it has been widely used as a cleaning fluid in the home and as a degreaser in industry. Because it is nonflammable, it was also used in fire extinguishers.

Nearly all carbon tetrachloride released to the environment exists in the atmosphere (73% is released to the atmosphere directly. Most of the carbon tetrachloride released to soil and water evaporates within a few days) (EPA 1991). As a result of releases to soil most carbon tetrachloride is expected to evaporate rapidly from soil due to its high vapor pressure (91.3 mm Hg). A smaller fraction of the carbon tetrachloride may adsorb to organic matter, since the octanol:water partition coefficient is about 110:1 ($\log K_{ow} = 2$) (Hansch and Leo 1979; Kenaga 1980). Due to the low adsorption of carbon tetrachloride in soil, leaching to groundwater is likely (Howard 1990), but is dependent somewhat on the organic carbon content of the soil. The retardation factor of carbon tetrachloride in breakthrough sampling in groundwater ranged from 1.4 to 1.7 indicating that soil adsorption is a relatively minor fate process and that carbon tetrachloride is moderately mobile in soil (Mackay et al. 1983). The log octanol/water partition coefficient ($\log K_{ow}$) of 2.64 for carbon tetrachloride (EPA 1984) suggests that bioaccumulation is at least possible under conditions of constant exposure and may occur in occupational settings or in people living at or near hazardous waste sites.

Chloroform

The major uses of chloroform are in fluorocarbon-22 (chlorodifluoromethane) and in the total production of refrigerants and fluoropolymers.

Based upon a vapor pressure of 159 mm Hg at 20 degrees Celsius, chloroform is expected to exist almost entirely in the vapor phase in the atmosphere (Boublik et al. 1984; Eisenreich et al. 1981). Large amounts of chloroform in the atmosphere may be removed by wet deposition since chloroform is highly soluble in water. Most of the chloroform removed in precipitation is likely to reenter the atmosphere by volatilization.

Photolysis and hydrolysis are not significant environmental fate processes for chloroform. Most of the chloroform released to the environment eventually enters the atmosphere, while much smaller amounts enter groundwater as a result of filtration through the soil (ATSDR 1993a).

Chloroform is highly mobile in soil with transport to groundwater likely. Volatilization of chloroform in near-surface soils is likely to an important removal mechanism. Transformation processes are significant in natural soils. Diffusion through soil-air pores up to the ground surface and subsequent removal by wind may be an important transport pathway. The mobility of chloroform in soil/groundwater systems is strongly influenced by the fraction expected to sorb to soil. Its low K_{ow} value (93) indicates weak absorption to soils or organic matter. Although it is likely to volatilize from soil surfaces, the fraction not subject to volatilization is expected to be mobile in groundwater (ADL 1986).

It is highly volatile and little potential for substantial bioaccumulation in living organisms. Chloroforms tend to specifically accumulate in the adipose tissue, which may have important consequences of bio-magnification in aquatic food chains.

Chloromethane

No information in the literature was found for the fate and transport of chloromethane.

Chrysene

See discussion for PAH.

Cyanide

Cyanides are used widely and extensively in the manufacture of synthetic fabrics and plastics, in electroplating baths and metal mining operations, as pesticidal agents and intermediates in agricultural chemical production, and in predator control devices. Anthropogenic sources of cyanide in the environment include certain industrial processes, laboratories, fumigation operations, cyanogenic drugs, fires, cigarette smoking, and chemical warfare.

Volatile cyanides occur only occasionally in the atmosphere, due largely to emissions from plating plants, fumigation, and other special operations (Towill et al. 1978). Under normal conditions cyanide has relatively low persistence in air, usually between 30 days and 1 year (Way 1981), although some atmospheric HCN may persist for up to 11 years (Marrs and Ballantyne 1987).

In water, cyanides occur as free hydrocyanic acid, simple cyanides, easily degradable complex cyanides such as $Zn(CN)_2$, and sparingly decomposable complex cyanides of iron or cobalt (Towill et al.

1978). Cyanide has relatively low persistence in surface water under normal conditions but may persist for extended periods in groundwater (Way 1981). Volatilization is the dominant mechanism for removal of free cyanide from concentrated solutions and is most effective under conditions of high temperatures, high dissolved oxygen levels, and increased concentrations of atmospheric carbon dioxide (Leduc et al. 1982). Loss of simple cyanides from the water column is primarily through sedimentation, microbial degradation, and volatilization (Leduc et al. 1982).

Cyanide seldom remains biologically available in soils because it is either complexed by trace metals, metabolized by various microorganisms, or lost through volatilization (Towill et al. 1978). Cyanide ions are not strongly adsorbed or retained in soils, and leaching into the surrounding groundwater will probably occur.

Decanal

No information in the literature was found for the fate and transport of decanal.

Dibenzofuran

No information in the literature was found for the fate and transport of dibenzofuran.

Dichlorodifluoromethane

No information in the literature was found for the fate and transport of dichlorodifluoromethane.

Di-2-ethylhexyl-phthalate

For the purposes of this data compilation, di-2-ethyl-phthalate is considered synonymous with di-2-ethylhexyl-phthalate. No information was found for a chemical called di-2-ethyl-phthalate but di-2-ethylhexyl-phthalate is a known chemical.

Di(2-ethylhexyl) phthalate (DEHP) is used as a plasticizer for polyvinyl chloride (PVC) and other polymers (Mannsville Chemical Corporation 1990) in large quantities and is likely to be released to air and water during production and waste disposal of these plastic products.

DEHP released to air will be carried for long distances in the troposphere and has been detected in air above oceans. Wash out by rain appears to be a significant removal process. It is unknown whether direct photolysis or photooxidation are important atmospheric process.

DEHP has a very low vapor pressure (6.45E-6 mm Hg at 25 °C; Howard et al. 1985) and a strong tendency to adsorb to soil and sediments. Experimental evidence demonstrates strong partitioning to clays and sediments with a corresponding K_{oc} reported to be near 1E+5. As a result, DEHP released to soil will neither evaporate nor leach into groundwater. Limited data is available to suggest that it may biodegrade in soil under aerobic conditions following microorganism acclimation.

DEHP in water will biodegrade (half-life 2-3 weeks) following a period of acclimation. It will also adsorb to sediments and bioconcentrate in aquatic organisms. DEHP has a low Henry's Law constant (1.1E-6 atm-m^3/mole) and is moderately soluble in water (0.3 mg/L at 25 °C). Evaporation and hydrolysis are not significant aquatic mechanisms for DEHP (Callahan 1979).

DEHP appears to rapidly biodegrade under aerobic conditions following acclimation in screening biodegradation tests in river or lake water, in water/sediment systems, and in activated sludge. However, in oligotrophic lake samples, no degradation occurs. Under anaerobic conditions in water/sediment samples, biodegradation appears to not occur. Limited data suggests some biodegradation in soil. DEHP abiotic photolysis in water has been estimated to have a half-life of 143 days.

DEHP does have a tendency to bioconcentrate in aquatic organisms (BCF has been measured at 100-10000 in fish and invertebrates).

Diethyl phthalate

Diethyl phthalate may have some natural sources since it has been detected in cranberries and as a volatile component of baked potatoes and roasted filberts; however, this may be the result of diethyl phthalate (DEP) use as a solvent in the preparation of these foods. Artificial sources for DEP include air emissions, aqueous effluent, and solid waste products from manufacturing and plastics processing. It is estimated that 0.5% of all DEP produced is lost during it manufacture from phthalic anhydride. Incineration of DEP-containing plastics releases DEP into the environment but waste disposal sites containing disposed plastics with DEP in them are the major reservoir of DEP in the environment. Volatilization and leaching from these materials are significant modes of transport of DEP into air, water, and soil.

If released to the atmosphere, DEP is expected to exist in the vapor form and be adsorbed to airborne particulates. DEP vapor is expected to react with photochemically generated hydroxyl radicals with an estimated reaction half-life of 22.2 hours at 25 °C. Physical removal by particulate settling and wash-out in precipitation is also likely.

If released to soil, DEP is expected to undergo aerobic biodegradation. Oxidation, chemical hydrolysis, and volatilization from wet soil surfaces are not expected to be significant fate processes. However, DEP may volatilize from dry soil. There is evidence that phthalate esters are slowly volatilized from plastics into air at high temperature suggesting that DEP may volatilize from dry soil surfaces. The K_{oc} value has been estimated to be as high as 526 and the common occurrence of DEP in bottom sediments indicate that adsorption to sediments may be significant and that DEP has low mobility in soil.

If released to water, DEP is expected to undergo aerobic biodegradation with half-lives ranging from about 2 days to more than 2 weeks. Anaerobic biodegradation is reported to occur much more slowly if at all. Removal of DEP by oxidation, chemical hydrolysis, direct and indirect photolysis, or bioaccumulation in aquatic organisms appears to be insignificant. The calculated Henry's Law constant of 4.8E-7 atm-m^3/mol suggests that volatilization of DEP would not be significant from most bodies of water with the exception of shallow rivers. Identification of DEP in dated sediment cores from the Chesapeake Bay indicates that DEP has accumulated and persisted in sediment for over a century.

Tests indicate DEP biodegrades in mixed microbial populations with half-lives ranging from 2.2 days to 28 days. Abiotic degradation appears significant with pH above 10. The measured bioconcentration factor for DEP in fish ranges from 15-117 as indicated by experiments. These BCF values indicate that bioaccumulation in aquatic organisms is not significant.

Dimethyl phthalate

No information in the literature was found for the fate and transport of dimethyl phthalate.

Di-n-butyl phthalate

Di-n-butyl phthalate is used primarily as a plasticizer for epoxy resins and polyvinyl chloride. It has been used in plastisol formulations for carpet back coating and other vinyl compounds. Di-n-butyl phthalate is also used as an adjusting agent for lead chromate pigments, as a concrete additive, as an insect repellent for the impregnation of clothing, as a solvent for perfume oils, and in polyvinyl acetate emulsions (Sax and Lewis 1987).

Although di-n-butyl phthalate has low volatility, it has been reported in both the vapor phase and as particulate in the atmosphere. In the air, di-n-butyl phthalate is transported from its origin and is subject to both wet (rain and snow) and dry (wind and settling) deposition on the earth's surface (Atlas and Giam 1981).

Although di-n-butyl phthalate is only poorly soluble in water, it may be transported in water following formation of chemical complexes between di-n-butyl phthalate and humic substances (Callahan et al. 1979). Adsorption onto soil and sediments appear to be a significant sink for di-n-butyl phthalate.

Most of the di-n-butylphthalate released into the environment is likely to sorb onto soil, with very little partitioning into air or water. Its high K_{ow} value (37,200) indicates relatively strong absorption to soils or organic matter. Vapor phase transport of di-n-butyl phthalate through air-filled soil pores is probably not significant.

Volatilization from surface soils is not likely. Its strong absorption to soils suggests that it will not be particularly mobile in groundwater (ADL 1986). In hazardous waste sites, the presence of common organic solvents such as alcohols and ketones may increase the solubility of relatively water insoluble compounds such as di-n-butyl phthalate, thus increasing the amount that may leach from the site into the subsoil and into the groundwater.

It is resistant to hydrolysis and direct photodegradation but is fairly readily biodegraded. Data indicate that di-n-butyl phthalate can be taken up by and accumulate in a variety of organisms.

Di-n-octylphthalate

Although relatively little specific information concerning di-n-octylphthalate is available, the environmental transport and fate of this contaminant can be largely inferred from data for phthalate esters as a group. Di-n-octylphthalate probably hydrolyzes in surface waters, but at such a slow rate that this process would not be significant under most conditions. Photolysis and oxidation do not appear to be important environmental fate processes (EPA 1985). No other information was found for the fate and transport of di-n-octylphthalate.

Ethanol

No information in the literature was found for the fate and transport of ethanol.

Ethylbenzene

Ethylbenzene is used primarily in the production of styrene (ACGIH 1986). Other uses of ethylbenzene include use as a solvent, as a constituent of asphalt and of naphtha, and in fuels (ACGIH 1986).

The physiochemical properties of ethylbenzene reveal a strong tendency for ethylbenzene to partition into the atmosphere (Mackay 1979). Depending upon site conditions, releases to the surface soil can result in substantial losses to the atmosphere in addition to subsurface infiltration. Vapor phase transport will occur from subsurface releases and during migration through the partitioning into air pockets within unsaturated soil pore spaces (Rhue et al. 1988)

Ethylbenzene is somewhat mobile in soil/water systems, especially in areas where adequate water is present. The log K_{ow} for ethylbenzene is 3.13. In surface soils. the majority of ethylbenzene present is expected to be sorbed to soil, with only a small fraction being lost via diffusion to deeper layers and volatilization. In deeper soils, a much higher fraction will be present in the soil-water phase. Migration of ethylbenzene through air-filled is likely to be an important transport mechanism. Ethylbenzene under normal conditions is not expected to undergo hydrolysis but can biodegrade if a sufficient microbial population exists (ADL 1987).

Fluoranthene

See discussion for PAH.

Fluorene

See discussion for PAH.

Formaldehyde

No information in the literature was found for the fate and transport of formaldehyde.

HMX (CYCLOTETRAMETHYLENETETRANITRAMINE)

Photolysis will degrade HMX in shallow surface water. HMX is practically insoluble in water and is nonhydroscopic. Its solubility in other solvents is comparable to that of RDX. HMX is considered extremely stable for a high explosive. HMX absorbs the greatest number of photons at wavelengths below 290 nanometers (nm) (Maycock et al. 1969); however, HMX degrades in natural light by absorbing light between 290 and 370 nm (Spanggord et al. 1983; Maycock et al. 1969; Smetana and Bulusu 1977). The photolytic decay rates for HMX range from 0.0036 to 0.4 (Zepp and Cline 1977), and the corresponding half-lives range from 1.7 to 192 days.

The information regarding HMX's tendency to sorb to soils is inconclusive. Data indicates adsorption of HMX is slight; however, leaching could be important (Burrows et al. 1989).

Bioaccumulation is probably not an important transport mechanism for HMX. Studies of the effects of oral doses of HMX (500 mg/kg) on mice and rats found that the chemical reached a peak concentration of 6 to 10 g/mL in plasma within 6 hours. In contrast, intravenous doses of HMX (2 mg/kg) reached a peak concentration of 0.5 to 1 g/mL within an hour (Wilson 1985). Aquatic BCFs can be calculated for explosives from empirical relationships, Burrows et al. (1989) calculated the HMX BCF in fish to be 0.49, which indicates only a very low tendency for HMX to accumulate in aquatic life.

Hydrazine

Hydrazine is a highly polar, weakly basic, fuming liquid that occurs naturally as a product of nitrogen fixation by Azotobacter agile. It is used as a rocket propellant, polymerization catalyst, a

blowing agent, a reducing agent, an oxygen scavenger in boiler water treatment, in the synthesis of maleic hydrazine, and in the manufacture of drugs. No other information was found for the fate and transport of hydrazine.

Indeno(1,2,3)pyrene

See discussion for PAH.

Iron

No information in the literature was found for the fate and transport of iron.

Isophorone

Isophorone is a solvent for a large number of natural and synthetic polymers, resins, waxes, fats, and oils. Specifically, it is used as a solvent for concentrated vinyl chloride/acetate-based coating systems for metal cans, other metal paints, nitrocellulose finishes, printing inks for plastics, some herbicide and pesticide formulations, and adhesives for plastics, poly(vinyl) chloride and polystyrene materials (Papa and Sherman 1981).

Based on its water solubility (12,000 ppm), some isophorone may wash out of the atmosphere; however, only limited amounts will be washed out because of the short atmospheric half-life of isophorone. Particularly during the day, when hydroxyl radical concentrations are highest, very little atmospheric transport will occur due to its fast reaction with hydroxyl radical.

In water, neither volatilization nor sorption to sediments is expected to be an important transport mechanism.

Bioconcentration of isophorone is not thought to be significant and possibly only one order of magnitude higher than concentrations in the surrounding area.

Methanol

No information in the literature was found for the fate and transport of methanol.

Methyl isobutyl ketone

No information in the literature was found for the fate and transport of methyl isobutyl ketone.

Methylene chloride (dichloromethane)

Methylene chloride is used as a solvent in paint strippers, as a propellant in aerosols, as a metal cleaning and finishing solvent, in electronics manufacturing, and as an agent in urethane foam blowing (Mannsville Chemical Products Corporation 1988).

Volatilization is an important removal process for methylene chloride in surface soils. Methylene chloride is expected to undergo rapid biotransformation and degradation due to hydrolysis. The half-life of methylene chloride volatilization from water has been found to be 21 minutes under experimental conditions (Dilling et al. 1975), but actual volatilization from natural waters will depend on the rate of mixing, wind speed, and other factors (Callahan et al. 1979). In deeper soil, most methylene chloride is

expected to be in the soil-water phase, moving freely with flowing groundwater. The persistence of methylene chloride in soil/groundwater systems can be as much as months or years (ADL 1987).

Methylene chloride is not strongly sorbed to soils or sediments (Dilling et al. 1975). Based on its low soil organic carbon partitioning coefficient of 25, methylene chloride is likely to be very highly mobile in soils (Bahnick and Doucette 1988) and can be expected to leach from soils into groundwater.

n-Propylbenzene

No information in the literature was found for the fate and transport of n-Propylbenzene.

Naphthalene

Naphthalene enters the atmosphere primarily from fugitive emissions and exhaust connected with production and use of fuel oil and gasoline. In addition, there are discharges on land and into water from spills during the storage, transport, and disposal of fuel oil and coal tar. Naphthalene is a component of crude oil and a product of natural uncontrolled combustion. The principal end use for naphthalene was the production of phthalic anhydride but was also used as a lubricant and in scintillation counting fluid. (EPA 1988b).

The sorption of naphthalene to soil will be low to moderate depending on the organic carbon content of the soil. Its passage through sandy soil will be rapid. It will undergo biodegradation, which may be rapid when the soil has been contaminated with other polycyclic aromatic hydrocarbons, with half lives ranging from a few hours to days, but slow otherwise (degradation half-life > 80 days). Evaporation of naphthalene from the top soil layer will likely occur, but decreases with depth.

Volatilization, photolysis, sorption, and biodegradation are the important loss mechanisms for naphthalene discharged into water. In slow moving water, biodegradation may be the most important process with half-life of 1-9 days. Removal through sediment sorption may be the dominant loss mechanism in slow moving waters with high concentrations of suspended solid, such as lakes and reservoirs. The photolysis half-life in near-surface water is about 3 days, but increases to over 500 days at depths of 5 m or more.

The most important process for the removal of naphthalene from the atmosphere is its reaction with photochemcially produced hydroxyl radicals and the half-life of this reaction is reportedly less than one day. In polluted urban air, reaction with NO_3 radicals may be an additional nighttime loss mechanism.

Naphthalene bioconcentrates to a moderate amount in fish and aquatic invertebrates; however, depurtation for most invertebrates when placed in naphthalene-free water is rapid and naphthalene is also readily metabolized in fish (Callahan 1979). People are commonly exposed to naphthalene from inhalation of ambient air, particularly in areas with heavy traffic and at gasoline filling stations. Spills on skin during automobile refilling may cause dermal exposure. Another source of inhalation exposure is tobacco smoke. Additional exposure in non-occupational scenarios may occur from the ingestion of naphthalene-contaminated drinking water and the consumption of contaminated foods. Various possibilities exist for occupational exposure, particularly through inhalation, in the chemical industry.

Nitrobenzene

The movement of nitrobenzene in soil, water and air is dominated by its water solubility (1900 ppm) (Verschueren 1983), moderate volatility, low octanol-water partition coefficient , and soil/sediment

sorption coefficient. There is no known mechanism of hydrolysis of nitrobenzene; however, photolysis and biodegradation are significant pathways (Callahan et al. 1979; Mabey et al. 1983). Photochemical oxidation of nitrobenzene by H_2O_2 yields p-, o-, and m-nitrophenols (Draper and Crosby 1984) with an estimated half-time of 250 days (Dorfman and Adams 1973). Direct photolysis, measured by Zepp and Scholtzhauer (1983), has a half-time of 2.5 to more than 6 days near the surface of bodies of water in the vicinity of 40°N latitude.

Jury et al. (1984) classified nitrobenzene as intermediately mobile, but noted that its loss from soil would be enhanced by evaporation of water. Sediment sorption and bioceoncentration into aquatic and terrestrial animals will be negligible, whereas plant uptake might be expected in terrestrial (McFarlane et al. 1987a, 1987b), but not aquatic systems (Geyer et al. 1984). Leaching through soil may be significant.

Conflicting results on the biodegradation of nitrobenzene are present. It has been shown to be almost completely removed from natural waters or in various sewage treatment processes in some studies. Other studies show the majority of nitrobenzene removed but at a much slower rate. A few studies have shown nitrobenzene to be either highly resistant to degradation or also inhibited biodegradation of other compounds in the medium.

Orthophosphate

No information in the literature was found for the fate and transport of orthophosphate.

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances. PAHs are ubiquitous in nature primarily as a result of natural processes such as forest fires, microbial and terrestrial vegetation synthesis, and volcanic activities (Eisler, 1987b). Some of the PAHs are used in medicines and others are used to make dyes, plastics, and pesticides. They are found throughout the environment in the air, water, and soil. Most PAHs do not occur alone in the environment, rather they are found as mixtures of two or more PAHs.

The molecular weight of the individual PAHs affects their mobility and solubility in the environment, with lower-weight compounds (acenaphthene, acenaphthylene, anthracene, fluorene, phenanthrene, fluoranthene, 2-methylnaphthalene, and pyrene) of more concern because they are generally more volatile and soluble than higher-weight compounds [benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene], which have strong sorption properties.

Of major environmental concern are mobile PAHs that vary in molecular weight from 128.16 (naphthalene) to 300.36 (coronene). Higher molecular weight PAHs are relatively immobile because of their large molecular volumes and their extremely low volatility and solubility.

When released to the atmosphere, PAH compounds will become associated with particulate materials. Their residence time in the atmosphere and transport to different geographic locations are governed by particle size, meteorological conditions, and atmospheric physics. Much of the PAHs released into the atmosphere eventually reach the soil by direct deposition or by deposition on vegetation.

In aquatic environments, PAHs may either evaporate, disperse into the water column, become incorporated into bottom sediments, concentrate in aquatic biota, or experience chemical oxidation and biodegradation (Suess 1976). PAH partitioning in sediments occurs in an equilibrium partitioning

process, with a potential for localized occurrences of high levels of dissolved PAHs (Edwards, 1983). PAHs will adsorb strongly onto suspended particulates and biota. The ultimate fate of those which have accumulated in the sediment is believed to be biodegraded and biotransformed by benthic organisms (EPA, 1980b).

In general, PAHs show little tendency to biomagnify, despite high lipid solubility, because most are rapidly metabolized by vertebrates. Plants and invertebrates, however, may bioaccumulate high concentrations of these compounds. Most studies conducted on PAHs are single compound laboratory tests which are inapplicable, for the most part, to field situations. Organism and species responses to these compounds are variable, and are largely affected by the presence of other inorganic and organic compounds (Eisler, 1987b).

Polychlorinated Biphenyls (PCBs)

PCBs, a group of synthetic halogenated aromatic hydrocarbons, were first prepared in 1881, and since 1930 have been in general use in products that include heat transfer agents, lubricants, dielectric agents, flame retardants, plasticizers, and waterproofing materials (Roberts et al. 1978). After 1971, they were used almost exclusively as insulating or cooling agents in closed electrical systems, such as transformers and capacitors (NAS 1979).

PCBs are a family of compounds that vary widely in physical, chemical, and biological properties. The fate of PCBs in the environment is affected by biodegradation and photolysis. Non-destructive processes that affect the distribution and transport of PCBs are absorption, volatilization, and bioaccumulation.

Volatilization and transport as an aerosol followed by fallout with dust or rain is the probable cause of the widespread distribution of PCBs (Callahan et al. 1979). The volatilization rate will be greater from soil with low organic carbon, due to the weaker sorption of PCBs (Shen and Tofflemire 1980). The volatilization rates also will be greater in moist soils due to the co-distillation of PCBs with water. Storm water runoff will also transport PCBs from soil to surface water.

In water, adsorption to sediments or other organic matter is a major fate process for PCBs. PCBs tend to have high octanol-water partitioning coefficients, low water solubilities, and sorb strongly to soil organic matter. Experimental and monitoring data have shown that PCB concentrations are higher in sediment and suspended matter than in the associated water column (Eisenreich et al. 1983).

PCBs are extremely stable compounds, and are slow to chemically degrade under environmental conditions. The tendency of PCBs to absorb to soil and hence their environmental persistence increases with the degree of chlorination as well as with the organic content of the soil. PCBs with a high degree of chlorination degrade very slowly in the environment. Significant leaching of PCBs from soil is not likely. The biota are another environmental compartment into which these compounds may be partitioned.

Phenanthrene

See discussion for PAH.

Phenol

Phenol, also called hydroxybenzene, is a common and important industrial chemical that enters the environment in wastewater and spills connected with its use in resins, plastics, adhesives, or other uses. It

is frequently found in wastewater from other commercial processes. Natural sources include animal wastes and decomposition of organic wastes. Other artificial sources include cigarette smoke, auto exhaust, disinfectants, and medicinal products. If released to the environment, biodegradation is the primary removal mechanism with half-life in the order of days.

If phenol is released to the atmosphere, it will exist predominantly in the vapor phase. Reaction with hydroxyl radicals in air has a half-life of about 0.6 days. At nighttime, phenol reacts with nitrate radicals with a reaction half-life determined by tests to be about 15 minutes. Phenol may be subject to removal in rain based on its detection in rainwater.

If phenol is released to soil, it will biodegrade in the soil rapidly (2-5 days). Degradation will also occur under anaerobic conditions but will be much slower. Despite its high solubility and poor adsorption on soil, biodegradation is sufficiently rapid that most groundwater is generally free of phenol. The exception is the case of large spills of high enough concentration to destroy the degrading microbial populations. Some evaporation and hydrolysis from surface spills is likely. Low adsorption to clay soils and silt loam have been reported (K_{oc} values of 39 and 91, respectively). Based on the calculated K_{oc} of 148, phenol should be relatively mobile in soil and may leach to groundwater.

If released to water, the primary removal process will be biodegradation which is generally be rapid; tests indicate degradation on the order of hours to weeks depending on the condition of the water. Degradation rates are much slower under anaerobic conditions. Photolysis is likely but not significant nor is evaporation of phenol in aquatic settings. However, phenols react relatively rapidly in sunlit natural water via reaction with photochemically produced hydroxyl and peroxyl radicals. Despite its moderate vapor pressure (0.524 mm Hg at 25 °C), phenol has a low Henry's Law constant (3.97E-7 atm-m^3/mol) and a low rate of evaporation from water. The estimated half-life from water is about 3.2 months.

Biodegradation in aerobic activated sludge has been tested to be better than 90% complete in 8 hours. Complete biodegradation in water has been observed in less than a day. Bioconcentration factors for aquatic organisms range from 1.9 to 277 suggesting phenol does not bioconcentrate significantly in aquatic organisms.

Propionitrile

No information in the literature was found for the fate and transport of propionitrile.

Pyrene

See discussion for PAH.

RDX (HEXAHYDRO-1,3,5-TRINITRO-1,3,5-TRIAZINE)

Transformation of RDX in the environment occurs most rapidly by photolysis. Spanggord et al. (1980) measured the degradation of RDX in distilled and filter-sterilized natural water in sunlight. They calculated a half-life of 13 days from their measurements of RDX disappearance from distilled water, 14 days in Holston River water, and 9 days in Searsville Pond water during cloudy January weather.

Hydrolysis should be a significant environmental fate of RDX only in sufficiently alkaline waters (Hoffsommer and Rosen 1973). Measurements of soil-water partition coefficient and organic-carbon partition coefficient (Tucker et al. 1985; Spanggord et al. 1980; and Sikka et al. 1980) indicate that RDX does not strongly adsorb to soils and sediments.

Biological processes can play a role in determining the fate of RDX. Microbial reduction of RDX usually tafes place under anaerobic conditions. McCormick et al. (1981) found that RDX disappeared from nutrient broth cultures within 4 days of anaerobic incubation, but found no loss from aerobic cultures or controls. Spanggord et al. (1980) found that aqueous solutions of RDX were aerobically degraded over 16 days from 10 ppm to 4 ppm by adapted microbes in Holston River water after a 20-day lag time. They reported that RDX does not undergo biotransformation without added nutrients or adapted bacteria.

In addition, studies by Cataldo et al. (1990) indicate that RDX bioaccumulation from soils by plants is a significant route of transport. Cataldo et al. (1990) amended soils, i.e., Burbank, Palouse, and Cinebar, with 10 ppm RDX and found that bush bean removed 55% of the RDX in Burbank soil, 37% from Palouse and 11% from Cinebar. In addition, blando brome grass was found to remove 45% the RDX amended to Burbank soil. From the Cataldo data a calculated BCF factor for Blando brome in Burbank soils is 91. Cataldo et al. (1990) also determined that partitioning of RDX occurs in plants; approximately 20% is stored in the roots and 80% among the shoots, fruits, leaves and seeds. These data suggest that bioaccumulation of RDX by plants presents a potential food-chain contamination route for RDX to higher trophic order organisms.

Sodium cyanide

No information in the literature was found for the fate and transport of sodium cyanide.

Sulfide

No information in the literature was found for the fate and transport of sulfide.

Sulfuric acid

No information in the literature was found for the fate and transport of sulfuric acid.

Terphenyl

No information in the literature was found for the fate and transport of terphenyl.

Tetrachloroethylene (PCE)

PCE is a commercially important chlorinated hydrocarbon solvent and chemical intermediate. It has been used as a dry cleaning and textile-processing solvent and for vapor degreasing in metal-cleaning operations. Other uses of PCE include nonflammable, recyclable dielectric fluid for power transformers, heat transfer medium, and intermediate the production of TCA (NIOSH 1978). PCE is not known to occur as a natural product (IARC 1979).

The relatively high vapor pressure (18.47 mm Hg at 25 °C) of PCE indicates that it exists entirely in the vapor phase in the ambient atmosphere and does not partition to atmospheric particulates. Significant evaporation from dry surfaces can also be predicted from the high vapor pressure. Physical removal from the atmosphere by means of wet deposition may be an important transport process for the compound. Dry deposition does not appear to be a significant removal process (Cupitt 1987).

PCE is relatively mobile in soil/groundwater systems, including transport of PCE vapors through air-filled soil pores. It is resistant to hydrolysis and biodegradation and, thus, may persist for months. Its

log K_{ow} value (2.60) indicates moderate adsorption to soils or organic matter. It is highly volatile, moderately sorbed to soil, and little potential for substantial bioaccumulation in living organisms. Although it is likely to volatilize from soil surfaces, the fraction not subject to volatilization is expected to be mobile in groundwater (ADL 1986).

Tetrahydrofuran

Tetrahydrofuran (THF) is used in large ($6.81*10^{10}$ grams/yr) quantities as a chemical intermediate and solvent. Because of its high vapor pressure and water solubility, significant amounts of the THF used as solvents will be released to the environment and workers will be exposed to it. Once released to the environment its behavior is not well understood and very little monitoring data are available. In the atmosphere, THF should degrade rapidly (half-life-hours to days), especially under smog situations and should be removed by rain. THF in water may biodegrade (only screening studies with sewage inoculum available) but acclimation is probably important. Evaporation should be very important (half-life 1.57 hr) but it will not be removed by photodegradation or adsorption to sediment. Spills on soil are expected to evaporate rapidly and leach into ground water. THF is not expected to bioconcentrate in fish or other aquatic organisms (Howard 1990).

Tetryl (2,4,6-Tetryl-Trinitophenylmethylnitramine)

Biodegradation and photolysis are the only significant degradation processes of tetryl. Kayser et al. (1984) reported that N-methylpicramide was the major detectable photoproduct of tetryl in distilled water exposed to laboratory "room light." Kayser et al. (1984) also stated that the photolysis rate is at least one more order of magnitude than the hydrolysis rate. Photolysis of tetryl in sunlight proceeds more slowly than the photolysis of the nitramines, requiring 20 days for completion. Hydrolysis has been demonstrated to occur to tetryl; however, this process is slow. Kayser et al. (1984) have reported a half-life (extrapolated) for this reaction of 302 ± 76 days at 20°C and a pH of 6.8. Kayser et al. (1984) also reported that the rate of hydrolysis increases with both pH and temperature.

Volatilization is considered to have an insignificant role in decreasing the concentration of explosives in environmental media (Burrows et al. 1989).

Adsorption to sediment is not an important environmental process for this explosive in surface water (Spanggord et al. 1980). No data on adsorption to soils was located, however, tetryl's low organic carbon partition coefficient (K_{oc}=44.67) indicates adsorption to soils is not a significant process. This is probably not an important transport process.

Based on a report by Trabalka and Garten (1982) on cutoff values for log octanol/water partition coefficient (K_{ow}) and solubility, tetryl does not accumulate in mammals or birds. This contention is supported by Burrows et al. (1989), who reported a tetryl bioconcentration factor (BCF) of 6.31 for fish and a very low BCF of 0.0023 for fat feed beef.

Toluene

The major use of nonisolated toluene (100%) is as a mixture added to gasoline (BTX) to improve octane ratings (EPA 1990). Nearly half of the isolated toluene which is not back-blended into gasoline is used to produce benzene (Fishbein 1988). Toluene is also used as a starting material in the synthesis of trinitrotoluene (TNT).

Although toluene is a liquid at room temperature, it is sufficiently volatile that the majority of toluene released to the environment partitions to air. Because toluene is moderately soluble in water (500 mg/L at 25 °C), it is likely that toluene is scrubbed from air by rainfall.

Toluene is relatively mobile in soil-water systems, and volatilization through air-filled pores is also possible. While toluene is resistant to hydrolysis, they are probably biodegradable. Toluene is expected to sorb primarily to soil in near-surface soils. In saturated, deeper soils, about half is likely to exist in the soil water phase. Its relatively low log K_{ow} value of 2.73 indicates weak absorption to soils or organic matter. Toluene is highly volatile from aqueous solutions, weakly absorbed by soil, and has a small potential to bioaccumulate in living organisms. Although it may volatilize from soil surfaces, the fraction not subject to volatilization may eventually migrate into groundwater (ADL 1987).

Total Petroleum Hydrocarbons (TPHs)

Petroleum is a combination of several products in varying amounts and combinations. Petroleum is composed of but is not limited to: Gasoline, Diesel, Fuel Oil No.2, Fuel Oil No.4, Kerosene, JP-4, JP-5, and Used Oil. Each of these products is a complex mixture of several hundred hydrocarbon compounds (PAHs, benzene, toluene, ethylbenzene, xylenes, ethylene dibromide, 1,2-dichloroethane, and methyl tert-butyl ether) and other additives (e.g., anti-knock agents, corrosion inhibitors, anti-oxidants, etc.). The actual composition of these products varies depending on the source, age, temperature, and other factors and conditions. Thus, no unique composition exists for any of the aforementioned products. The behavior of these products in the environment depends on the properties of the individual constituents and their concentrations (State of Idaho 1996).

The TRV for benzene was used for TPH and is thought to have similar fate and transport properties.

Tributyl phosphate

No information in the literature was found for the fate and transport of tributyl phosphate.

Trichloroethylene (TCE)

The major use of trichloroethylene (80% of consumption) is as a solvent for the vapor degreasing of fabricated metal parts. TCE has a variety of solvent applications as well as being used as a chemical intermediate. There are no known natural sources of trichloroethylene.

Trichloroethylene released to the atmosphere will exist primarily in the vapor phase based on its relatively high vapor pressure. It will react fairly rapidly, especially under smog conditions, in the atmosphere with residence times of around 5 days.

Trichloroethylene will also leach to groundwater rapidly. TCE is relatively mobile in soil/groundwater systems, particularly those with low organic matter content. Volatilization of TCE from surface soils can be significant. Spills or other releases of trichloroethylene on soil will result in rapid evaporation due to the high vapor pressure of trichloroethylene. Transformation of TCE via biodegradation and hydrolysis are not expected to be substantial in natural soils. Most TCE released onto surface soils will volatilize. TCE percolating through the soil in minimally retarded (ADL 1987). Most of the TCE present in surface soils will volatilize. Diffusion through soil-air pores up to the ground surface and subsequent removal by wind may be an important removal pathway. The mobility of TCE in soil/groundwater systems is strongly influenced by the fraction expected to sorb to soil. Its relatively low

log K_{ow} value (2.42) indicates fairly weak absorption to soils or organic matter. TCE is highly volatile in aqueous systems, moderately soluble in water, weakly absorbed to soil or organic matter, and not expected to substantially bioaccumulate in living organisms. The primary removal process for aquatic releases of trichloroethylene is evaporation. Half-life for aquatic evaporation of trichloroethylene ranges from minutes to hours depending on turbulence. Biodegradation, hydrolysis, and photooxidation are extremely slow by comparison. Though biodegradation in groundwater has been inferred, hydrolysis appears not to be an important process for terrestrial spills of trichloroethylene (ADL 1987).

Trimethylopropane-triester

No information in the literature was found for the fate and transport of trimethylopropane-triester.

Vinyl acetate

No information in the literature was found for the fate and transport of vinyl acetate.

Xylene

Fate and transport information was found for three xylenes: 1,2-xylene (CAS 95-47-6), also called o-xylene; 1,3-xylene (CAS 108-38-3), also called m-xylene; and 1,4-xylene (CAS 106-42-3), also known as p-xylene. The fate and transport are essentially the same for all three and are summarized below.

Xylenes enter the atmosphere primarily from fugitive emissions and exhaust connected with their use in gasoline. Industrial sources include emissions from petroleum refining, coal tar and coal gas distillation, and from their use as solvents (HSBD 1988). Discharges and spills on land and in waterways result from xylene use in diesel fuel and gasoline. Natural sources of xylenes include coal tar, petroleum, forest fires, and plant volatiles.

Most of the xylene released into the atmosphere photochemically degrades by reaction with hydroxyl radicals (half-life 1.5 -15 hr for o-xylene, 1.0-10 hr for m-xylene, 1.7-18 hr for p-xylene). Xylenes are also typically scavenged from the atmosphere by rainfall.

When spilled on land, xylenes volatilize and leach into the ground. Xylenes are moderately mobile in soil and may leach into groundwater where they have been known to be detectable for several years, although there is evidence that xylenes biodegrade in both soil and groundwater. This may be because xylenes degrade under aerobic conditions and denitrifying conditions appear to be required when oxygen is lacking. No measured K_d or K_{oc} values have been determined for xylenes; however, low to moderate adsorption would be expected based on their octanol/water partition coefficients.

The dominant removal process in water is volatilization (half-life 1-5.5 days). Xylenes have Henry's Law constants on the order of 1E-3 atm-m3/mole, which indicates rapid volatilization. Some adsorption to sediment will occur. Although xylenes are biodegradable and have been observed to biodegrade in seawater, there is insufficient data to assess the rate of this process in surface waters.

Standard biodegradation tests have been conducted on xylenes using sewage, activated sludge, and seawater with various inocula. Tests showed complete degradation within 8 days for p-xylene in groundwater. Other tests indicate up to 55 mg/day biodegradation of xylenes in shallow unconfined aquifers when oxygen is present. Decreased oxygen resulted in lower biodegradation rates. Under anaerobic conditions, several months are necessary for microorganisms to produce the necessary enzymes for the necessary denitrification; subsequent biodegradation was then rapid.

Bioconcentration is not expected to be significant. Xylene bioconcentration has not been observed much. The BCF has been measured as 1.33 and 0.79 for eels and clams and estimated at 2.12 for fish in general. The primary source of exposure to humans is from the air, especially where emissions from petroleum products or motor vehicles are high or solvents containing p-xylene are used.